United States Air Force
611th Air Support Group/
Civil Engineering Squadron

Elmendorf AFB, Alaska

Final
Risk Assessment

Wainwright Radar Installation,
Alaska

Prepared by:

ICF Technology Incorporated

18 JANUARY 1996

PREFACE

This report presents the findings of Risk Assessments at sites located at the Wainwright radar installation in northern Alaska. The sites were characterized based on sampling and analyses conducted during Remedial Investigation activities performed during August and September 1993. This report was prepared by ICF Technology Incorporated.

This report was prepared between January 1995 and January 1996. Mr. Samer Karmi of the Air Force Center for Environmental Excellence was the Alaska Restoration Team Chief for this task. Dr. Jerome Madden and Mr. Richard Borsetti of the 611 CES/CEVR were the Remedial Project Managers for this project.

Approved:

Thomas McKinney
Program Director
ICF Technology Incorporated

THIS PAGE INTENTIONALLY LEFT BLANK

NOTICE

This report has been prepared for the United States Air Force (Air Force) by ICF Technology Incorporated for the purpose of aiding in the implementation of final remedial actions under the Air Force Installation Restoration Program (IRP). As the report relates to actual or possible releases of potentially hazardous substances, its release prior to an Air Force final decision on remedial action may be in the public's interest. The limited objectives of this report and the ongoing nature of the IRP, along with the evolving knowledge of site conditions and chemical effects on the environment and health, must be considered when evaluating this report, since subsequent facts may become known which may make this report premature or inaccurate. Acceptance does not mean that the United States Air Force adopts the conclusions, recommendations or other views expressed herein, which are those of the contractor only and do not necessarily reflect the official position of the United States Air Force.

Government agencies and their contractors registered with the Defense Technical Information Center (DTIC) should direct requests for copies of this report to: DTIC, Cameron Station, Alexandria, Virginia 22304-6145.

Non-Government agencies may purchase copies of this document from: National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, Virginia 22161.

THIS PAGE INTENTIONALLY LEFT BLANK

TABLE OF CONTENTS

TABLE OF CONTENTS (CONTINUED)

TABLE OF CONTENTS (CONTINUED)

TABLE OF CONTENTS (CONTINUED)

TABLE OF CONTENTS (CONTINUED)

APPENDICES

LIST OF TABLES

LIST OF TABLES (CONTINUED)

LIST OF FIGURES

LIST OF ACRONYMS AND ABBREVIATIONS

ADD	Average Daily Dose
ADEC	Alaska Department of Environmental Conservation
Air Force	United States Air Force
API	American Petroleum Institute
ARAR	Applicable or Relevant and Appropriate Requirements
AWQC	Ambient Water Quality Criterion
BCF	Bioconcentration Factors
BTEX	Benzene, Toluene, Ethylbenzene, and Xylene
CDI	Chronic Daily Intake
COC	Chemical of Concern
DEW	Distant Early Warning
DRPH	Diesel Range Petroleum Hydrocarbons
ECAO	Environmental Criterion Assessment Office
EPA	U.S. Environmental Protection Agency
ERA	Ecological Risk Assessment
GRPH	Gasoline Range Petroleum Hydrocarbons
HEAST	Health Effects Assessment Summary Tables
HQ	Hazard Quotient
HSDB	Hazardous Substance Data Bank
HVOC	Halogenated Volatile Organic Compound
IRIS	Integrated Risk Information System
IRP	Installation Restoration Program
IS	Onsite Dietary Intake
LADD	Lifetime Average Daily Dose

LIST OF ACRONYMS AND ABBREVIATIONS (CONTINUED)

LOEL	Lowest Observed Effect Level
MDEP	Massachusetts Department of Environmental Protection
MOGAS	Motor Vehicle Gasoline
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
PAHs	Polynuclear Aromatic Hydrocarbons
PCB	Polychlorinated Biphenyl
RBSL	Risk-Based Screening Level
RfD	Reference Dose
RI/FS	Remedial Investigation/Feasibility Study
RME	Reasonable Maximum Exposure
RI	Remedial Investigation
RRPH	Residual Range Petroleum Hydrocarbons
SF	Slope Factor
SIF	Scaling Factor
SRR	Short Range Radar
SVOC	Semivolatile Organic Compound
TPH	Total Petroleum Hydrocarbon
TRV	Toxicity Reference Value
UCL	Upper Confidence Limit
UF	Uncertainty factor
USFWS	U.S. Fish and Wildlife Service
UST	Underground Storage Tank
VOC	Volatile Organic Compound

1.0 INTRODUCTION

This document contains the baseline human health risk assessment and the ecological risk assessment (ERA) for the Wainwright Distant Early Warning (DEW) Line radar installation. Six sites at the Wainwright radar installation underwent remedial investigations (RIs) during the summer of 1993. The presence of chemical contamination in the soil, sediments, and surface water at the installation was evaluated and reported in the Wainwright Remedial Investigation/Feasibility Study (RI/FS) (U.S. Air Force 1996). The analytical data reported in the RI/FS form the basis for the human health and ecological risk assessments. The primary chemicals of concern (COCs) at the six sites are diesel and gasoline from past spills and/or leaks. The general location of the Wainwright radar installation is shown in Figure 1-1. The six sites investigated and the types of samples collected at each site are presented in Table 1-1.

The purpose of the risk assessment is to evaluate the human and ecological health risks that may be associated with chemicals released to the environment at the six sites investigated during the RI. The risk assessment characterizes the probability that measured concentrations of hazardous chemical substances will cause adverse effects in humans or the environment in the absence of remediation. The risk assessment will be used to determine if remediation (site cleanup) is necessary and, if so, to rank sites for remedial action.

1.1 ORGANIZATION OF REPORT

Section 1.0 contains introductory information regarding the installation location and conditions, and a summary outline of the approach to the human health and ecological risk assessments. Section 2.0 is the Baseline Human Health Risk Assessment, and Section 3.0 is the Ecological Risk Assessment. References are presented in Section 4.0. Section 2.0, Baseline Human Health Risk Assessment, is composed of:

- **Selection of Site Contaminants**. Presents the COCs for human health and describes how they were selected for this risk assessment.

- **Exposure Assessment**. Identifies the pathways by which potential human exposures could occur, and estimates the magnitude, frequency, and duration of those exposures.

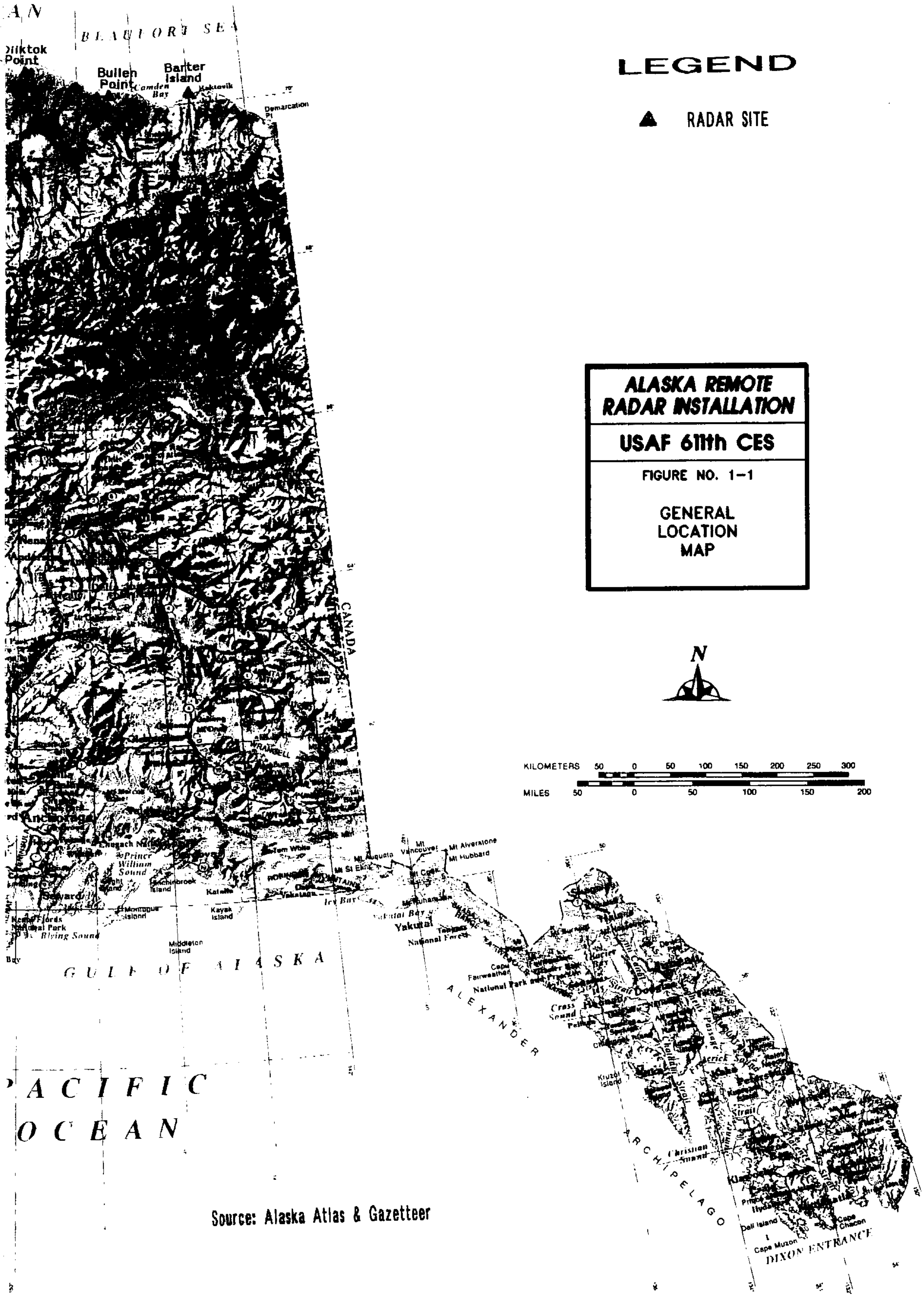
- **Toxicity Assessment**. Summarizes the toxicity of the selected COCs and the relationship between magnitude of exposure and the development of adverse health effects.

- **Risk Characterization**. Integrates the toxicity and exposure assessments to estimate the potential risks to human health from exposure to chemicals in environmental media.

THIS PAGE INTENTIONALLY LEFT BLANK


ARCTIC OCEAN
BEAUFORT SEA
Barrow
Point Barrow
Wainwright
Point Lonely
Oliktok Point
Bullen Point
Barter Island
Point Lay
Cape Lisburne
Point Hope
CHUKCHI SEA
RUSSIA
UNITED STATES
Kivalina
Kotzebue
KOTZEBUE SOUND
BERING STRAIT
Shishmaref
Cape Espenberg
Big Diomede Island
Little Diomede Island
King Island
Port Clarence
Nome
Gambell
Savoonga
SAINT LAWRENCE ISLAND
Norton Bay
NORTON SOUND
Stuart Island
Stebbins
BERING SEA
Emmonak
Alakanuk
Cape Romanzof
Hooper Bay
Saint Matthew Island
Nelson Island
Nunivak Island
Kipnuk
Quinhagak
KUSKOKWIM BAY
Goodnews Bay
Platinum
Cape Newenham
Togiak Bay
Hagemeister Island
Cape Constantine
BRISTOL BAY
Saint Paul Island
Saint Paul
PRIBILOF ISLANDS
Saint George
Saint George Island
Kvichak Bay
Iliamna Lake
Augustine Vol
Anchorage
Kenai
Soldotna
Homer
Seldovia
Anchor Point
Seward
Prince William Sound
Montague Island
Kayak Island
Middleton Island
Kennedy Entrance
Barren Islands
Stevenson Entrance
SHELIKOF STRAIT
Kodiak
Trinity Islands
GULF OF ALASKA
PACIFIC OCEAN
Pilot Point
Port Heiden
Herendeen Bay
Chignik Bay
Sutwik Island
Semidi Islands
Chirikof Island
Stepovak Bay
Sand Point
Shumagin Islands
King Cove
Cold Bay
Izembek National Wildlife Refuge
Unimak Island
Unimak Pass
Sanak Islands

Source: Alas



ALASKA REMOTE RADAR INSTALLATION

USAF 611th CES

FIGURE NO. 1-1

GENERAL LOCATION MAP

Source: Alaska Atlas & Gazetteer

TABLE 1-1. SITES EVALUATED AT WAINWRIGHT DEW LINE INSTALLATION

SITE NAME	SITE ID NUMBER	SOIL	SEDIMENTS	SURFACE WATER
Drum Storage Area	ST02	X	NA	NA
Diesel Fuel Spills	SS04	X	X	X
Landfill	LF05	X	X	X
Garage	SS07	X	X	X
Airstrip Diesel	SS08	NA	X	X
Vehicle Storage Area	SS09	X	X	X

X Chemical analyses were performed on these media.
NA No chemical analysis was performed.

- **Risk Assessment Uncertainty**. Describes the potential shortcomings in the data and the methods used to develop the risk assessment, and the uncertainties in the interpretation of the data and the risk characterization results.

Section 3.0, the Ecological Risk Assessment, is composed of:

- **Selection of Site Contaminants**. Presents the COCs for ecological receptors and describes how they were selected for the ERA.

- **Ecological Exposure Assessment**. Identifies the potential receptors and representative species, habitat suitability, and exposure pathways.

- **Ecological Toxicity Assessment**. Describes the potential effects of site contaminants on the representative species.

- **Risk Characterization for Ecological Receptors**. Evaluates the likelihood of adverse effects on ecological receptors.

- **Ecological Uncertainty Analysis**. Describes the potential shortcomings in the data and the methods used to develop the ERA, and the uncertainties in the interpretation of the data and the ecological risk characterization results.

- **Summary of Ecological Risks**. Presents a summary of ecological risks associated with the six sites at the Wainwright DEW Line installation.

Appendix A contains the human health risk assessment spreadsheets used to estimate chemical intake, noncancer hazard, and excess lifetime cancer risk. Appendix B consists of toxicology profiles. Exposure calculations and equations for ecological receptors are presented in Appendix C through F. Appendix G contains the RI analytical data for all sites from which the COCs were selected and upon which the human health and ecological risk assessments are based.

1.2 RISK ASSESSMENT GUIDANCE DOCUMENTS

The following guidance documents were used to develop the human health and ecological risk assessments:

- *Risk Assessment Guidance for Superfund: Volume 1, Human Health Evaluation Manual (Part A)* [U.S. Environmental Protection Agency (EPA) 1989a];

- Region 10 Supplemental Risk Assessment Guidance for Superfund (EPA 1991a);

- *Risk Assessment Guidance for Superfund: Volume 2, Environmental Evaluation Manual* (EPA 1989b);

- General Guidance for Ecological Risk Assessment at Air Force Bases (MITRE 1990);

- Handbook to Support the Installation Restoration Program (IRP) Statements of Work (U.S. Air Force 1991); and

- Framework for Ecological Risk Assessment (EPA 1992a).

1.3 INSTALLATION DESCRIPTION AND ENVIRONMENTAL SETTING

The Wainwright radar installation is located at the mouth of the Kuk River on the Chukchi Sea. The installation was constructed as an auxiliary station in 1953 and deactivated in 1989. The community of Wainwright is located approximately 4 miles from the installation and had a population of 584 in 1993, of which 90 percent was Inupiat (North Slope Eskimo) (Harcharek 1994).

The installation is located on 1,191 acres owned by the United States Air Force (Air Force). The Short Range Radar (SRR) system under construction in 1992 was operational in 1994. The inactive portion of the installation consists of one module train, rotating radar, and supporting facilities. The module train housed the electric equipment work areas, the radar tower, a power plant, a limited number of personnel quarters, administration offices, a mechanical room with emergency boiler and fuel storage, and dining, kitchen, and recreation areas. The installation has minimal aircraft support facilities, including a runway of 3,500 feet. There are no oil and gas exploration or production activities in the area.

The active SRR system that was constructed in the vicinity of the Vehicle Storage Area (SS09) consists of a radar tower, technical services building, and the satellite ground terminals.

The general location of the installation is shown in Figure 1-1. An area location map is presented in Figure 1-2, and a site plan of the installation is shown in Figure 1-3.

1.3.1 Drum Storage Area (ST02)

The Drum Storage Area (ST02) site is a gravel pad located southwest of the main station adjacent to the lagoon at the end of the access road. Approximately fifteen 55-gallon drums are present at the site. Most of the drums at the site are empty; others contain rainwater. A platform support structure exists at the south end of the site, and solidified bags of concrete and wood debris remain along the beach and at the north end of the site. The site was used for temporary storage of drummed products. Campfire ashes located in the middle of the gravel pad indicate the site may have been used by the residents of Wainwright.

1.3.2 Diesel Fuel Spills (SS04)

The Diesel Fuel Spills (SS04) site consists of soil/sediment and a gravel pad located below and adjacent to the west end of the module train. Two 10,000-gallon fuel spills were reported at the powerhouse section of the module train in the 1970s. Approximately 4,000 gallons from the second spill were recovered and reused. The spills have been estimated to extend from the midpoint of the module train to the western edge of the gravel pad. Smaller spills may have resulted from transfers of diesel oil from bulk fuel storage to the module train day tanks.

1.3.3 Landfill (LF05)

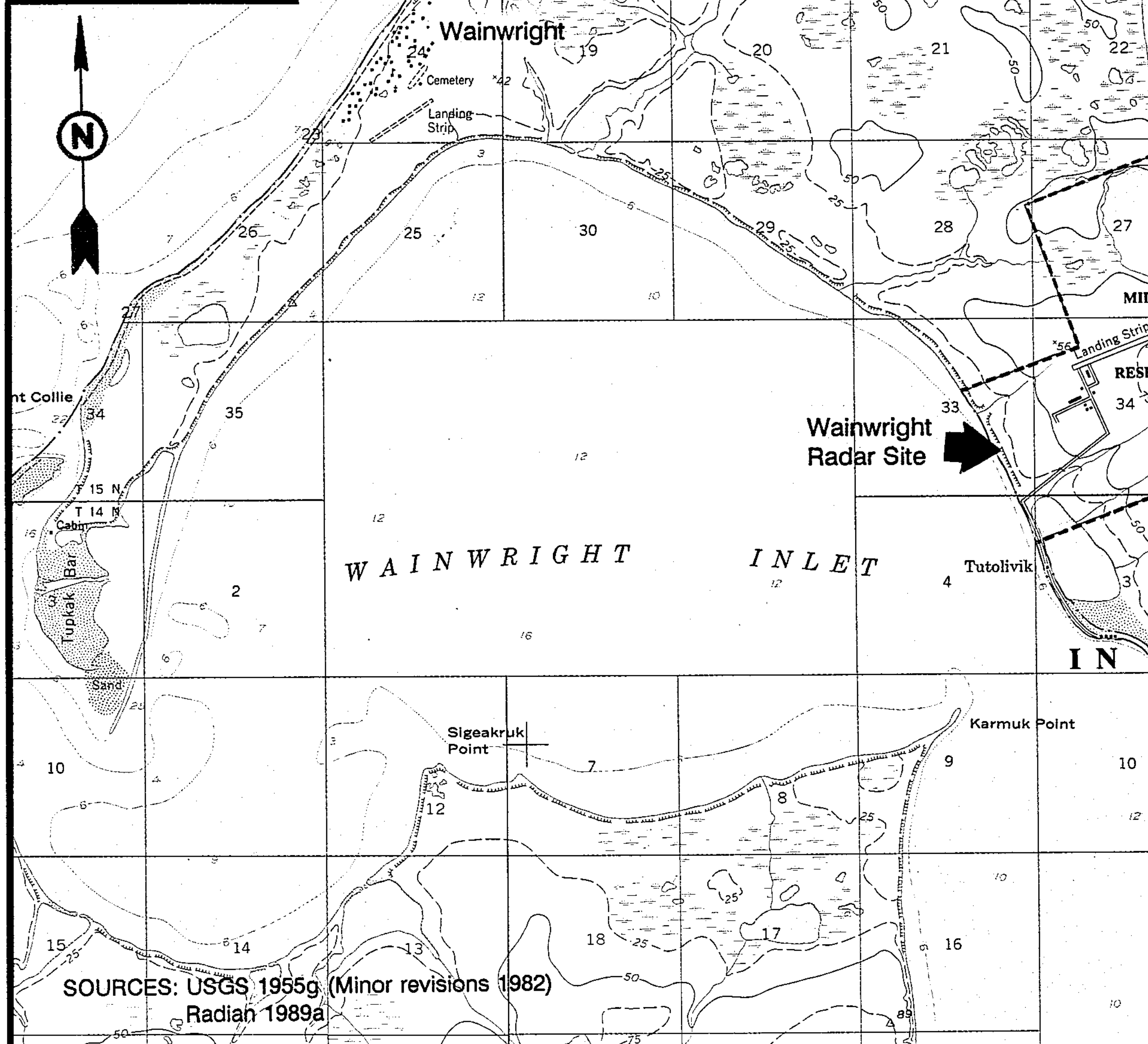
The Landfill (LF05) site is located on the tundra, which gently slopes to the Kuk River, approximately 800 feet southwest of the motor vehicle gasoline (MOGAS) tanks on the south end of the main station area. The inactive Landfill covers approximately half of an acre and is covered with gravel to a depth of approximately four feet. The Landfill received all wastes generated at the station from approximately 1974 to 1989.

1.3.4 Garage (SS07)

The Garage (SS07) site is located north of the module train and west of the warehouse. The Garage building is approximately 80 feet by 40 feet and was used for vehicle maintenance and storage. The building is raised approximately four feet above the tundra and is bounded by a four foot gravel pad on all sides. Floor drains in this building previously discharged directly to the tundra; however, the site has been inactive since 1989. Culverts lead from under the Garage to the tundra to the west and to a large ponded area surrounded by a gravel berm to the east.

THIS PAGE INTENTIONALLY LEFT BLANK

DRAWING No. WAI-AREA



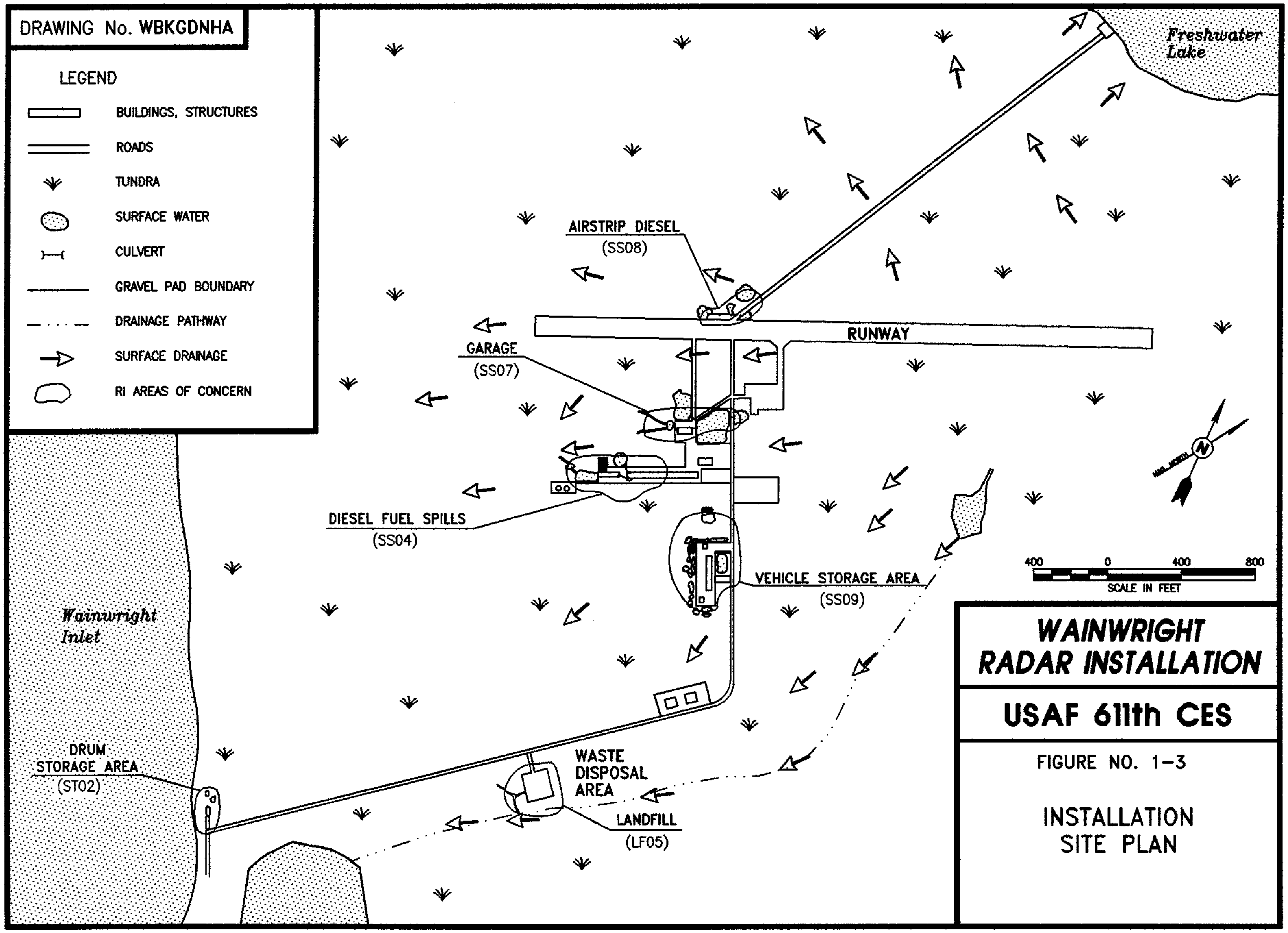
WAINWRIGHT RADAR INSTALLATION

USAF 611th CES

FIGURE NO. 1-2

AREA LOCATION MAP

THIS PAGE INTENTIONALLY LEFT BLANK



DRAWING No. WBKGDNHA
LEGEND
BUILDINGS, STRUCTURES
ROADS
TUNDRA
SURFACE WATER
CULVERT
GRAVEL PAD BOUNDARY
DRAINAGE PATHWAY
SURFACE DRAINAGE
RI AREAS OF CONCERN
Freshwater Lake
AIRSTRIP DIESEL (SS08)
RUNWAY
GARAGE (SS07)
DIESEL FUEL SPILLS (SS04)
VEHICLE STORAGE AREA (SS09)
Wainwright Inlet
DRUM STORAGE AREA (ST02)
WASTE DISPOSAL AREA
LANDFILL (LF05)
400 0 400 800
SCALE IN FEET
WAINWRIGHT RADAR INSTALLATION
USAF 611th CES
FIGURE NO. 1-3
INSTALLATION SITE PLAN

THIS PAGE INTENTIONALLY LEFT BLANK

1.3.5 Airstrip Diesel (SS08)

The Airstrip Diesel (SS08) site is located adjacent to the north side of the airstrip at the junction of the road to Freshwater Lake. The area consists of tundra and a gravel pad elevated approximately four feet above the adjacent tundra. A helicopter pad was under construction during the 1993 field sampling season at this site. No documentation of spills has been found for this site.

1.3.6 Vehicle Storage Area (SS09)

The Vehicle Storage Area (SS09) site consists of a gravel pad that was historically used for vehicle storage. The site is approximately 100 yards southeast of the module train in the vicinity of the new SRR system. New construction on, and adjacent to, the site includes the SRR tower, a technical services building, and two satellite ground terminals. A gravel pad was added to the original Vehicle Storage Area and adjacent road during construction of the SRR structures. During the current construction activities, soil boring materials that were considered potentially contaminated were stockpiled north of the Vehicle Storage Area site. These stockpiled soils were sampled as part of the RI at the site.

1.4 APPROACH TO HUMAN HEALTH RISK ASSESSMENT

The Wainwright DEW Line installation presents a unique challenge in development of a human health risk assessment. Many of the conventional assumptions applied in risk assessments do not apply to the North Slope of Alaska. Wainwright is remote and sparsely populated. Native residents, largely Inupiats, follow a lifestyle that includes a significant subsistence component; much of their food consists of mammals (whales, seals, moose, and caribou), aquatic life (arctic char), and birds (ptarmigan and ducks) that are abundant in this area of the arctic. The climate is generally harsh, and the soil and surface water are frozen for approximately nine months of the year.

The general approach to the human health risk assessment was to quantify the excess lifetime cancer risk or the noncancer hazard for the site contaminants detected at each of the six sites at the installation. The maximum concentration of each chemical detected was used instead of an arithmetic mean or 95th percentile upper confidence limit (UCL) because contamination was detected infrequently and found to be generally of low concentration. Incorporating nondetects into the calculation of an average or UCL when the frequency of positive detects is low tends to yield low and unreliable estimates of contamination. Use of the maximum concentration yields a conservative estimate of risk or hazard.

To the extent possible, site-specific information was incorporated into the development of the exposure assumptions. The harsh climate naturally serves to limit exposure to contaminated soil, sediment, and surface water.

Residential exposure assumptions were used to reflect the upper-bound potential future risk. Several North Slope communities have requested use of inactive buildings at DEW Line

installations; therefore, an evaluation using potential residential scenarios at the installations and sites was conducted.

Excess lifetime cancer risk and noncancer hazard were calculated for the soil/sediment ingestion and water ingestion pathways. Other pathways were eliminated from consideration as described in Section 2.2, the Human Health Risk Exposure Assessment.

1.5 APPROACH TO ECOLOGICAL RISK ASSESSMENT

The objective of the ERA is to estimate potential impacts to aquatic and terrestrial plants and animals at the Wainwright radar installation. The MITRE guidance (1990) suggests that ERAs should "estimate the potential for occurrence of adverse effects that are manifested as changes in the diversity, health and behavior" of ecosystems. MITRE proposes that this can be accomplished by:

- Estimating the health risk to individual species;
- Evaluating the health of the community of exposed species; and
- Determining the potential adverse effects of contamination over several life cycles of the species under study.

Because this is a screening level assessment, the scope of this ERA is limited to the first task: estimating the risk to individual species. If a potential risk to individual species was identified, further work may be recommended to evaluate the community and life cycle effects. It is important to note that the health risk to an individual species is different from the health risk to an individual within a species. The former refers to population level biology, where the individual is not considered a relevant endpoint. The latter assesses the risks to an individual. In this assessment, the individual is considered only in the case of threatened or endangered species.

2.0 BASELINE HUMAN HEALTH RISK ASSESSMENT

The purpose of the baseline human health risk assessment for the Wainwright DEW Line installation is to provide a basis for developing a risk management plan, including remedial action alternatives based on data from the RI/FS. The risk assessment develops numerical estimates of cancer risk and noncancer hazard for each site where sufficient information is available. Where information is not adequate to quantify noncancer hazard or cancer risk for a given COC, a qualitative discussion of the toxicity of that COC is provided in the Toxicity Profiles (Appendix B).

This risk assessment addresses issues unique to this location as described in the introduction. It follows the conventional approach in that it is comprised of six sections:

- Identification of COCs - in which the chemicals detected in environmental samples are compared to risk-based screening levels (RBSLs) and concentrations considered to be applicable or relevant and appropriate requirements (ARARs);

- Exposure assessment - in which the frequency, duration, and magnitude of potential exposures to the COCs are estimated;

- Toxicity assessment - in which the toxicology of the COCs is assessed;

- Risk characterization - in which the potential for adverse health effects in humans as a result of exposure to the COCs is quantified (as appropriate) or qualitatively discussed;

- Uncertainty assessment - in which the general sources of uncertainty in the risk assessment process and the site-specific sources of uncertainty are discussed; and

- Risk assessment summary and conclusions - in which the human health risks at each of the sites are summarized and conclusions based on these risks are presented.

2.1 IDENTIFICATION OF CHEMICALS OF CONCERN

COCs for human health were selected for each site at the Wainwright installation based on comparison of chemical concentrations to RBSLs, naturally-occurring background concentrations, ARARs, and safe levels of essential human nutrients (e.g., calcium, magnesium, sodium, and potassium).

This section discusses the RI sampling strategy and an evaluation of data prior to screening (Section 2.1.1), describes and presents equations for calculating RBSLs (Section 2.1.2), identifies

chemicals that are essential human nutrients (Section 2.1.3), describes the collection of background samples (Section 2.1.4), and discusses the selection of COCs (Section 2.1.5).

2.1.1 Sampling Strategy and Evaluation of Analytical Data

The RI sampling strategy at the Wainwright sites was to characterize the nature and extent of potential contamination at each site. Suspected source areas were sampled to determine the concentrations of contaminants, if any, at the areas likely to have the highest concentrations. Migration pathways from the source areas were sampled to determine the extent, if any, that the contaminants had migrated from the sites. If no discernable pathways were evident, an attempt was made to sample around the source areas to determine the extent of site contaminants. Quick turn-around analyses were conducted on samples from the first sampling event, and a second round of sampling was conducted at those sites where further characterization of the nature and extent of contamination was needed.

Sample types included surface and subsurface soil/sediment samples and surface water samples. In almost all cases, samples were discrete grab samples from one sample location. Surface soil and sediment samples were collected in gravel and tundra areas at or near the ground surface (from ground surface to approximately six inches in depth). Subsurface soil samples were mainly collected in gravel pad areas where unsaturated conditions allowed vertical migration of contaminants. Sediment samples were collected below shallow ponds or streams, or in areas that visually appeared to have been previously covered with water. Surface water samples were collected from ponds, streams, springs, or leachate areas. Surface water samples underwent both total and dissolved metal analyses; however, the total metal analytical results were used in the risk assessment. A summary of the 1993 RI sampling and analyses conducted at the installation is presented in Appendix G.

Before screening for COCs, the results of the RI sampling program were sorted by medium (i.e., soil, sediment, and surface water) and reviewed for quality. The review included an evaluation of the analytical methods used, the sample quantitation limits, and qualified data, and a comparison to background levels and laboratory and field blanks. Analytical data were reviewed for completeness, comparability, representativeness, precision, and accuracy. In addition, data validation qualifiers were considered in assessing the quality of the data. The review and validation of analytical data determined that a minimal amount of data was not usable. These data were qualified with an "R" and were not used in the risk assessment.

As outlined in the Risk Assessment Guidance for Superfund (EPA 1989a), site data were compared to available blank (laboratory, field, and trip) data. The data from blanks are presented in Appendix G. In accordance with EPA (1989a), if the detected concentration in a sample was less than 10 times the concentration from blanks for common laboratory contaminants (e.g., acetone, 2-butane, methylene chloride, toluene, and the phthalate esters) the chemical was not selected for evaluation in the risk assessment. For those organic or inorganic chemicals that are not considered by EPA to be common laboratory contaminants (all other compounds), if the detected concentration was less then five times the maximum concentration detected in the blanks, the chemical was not selected for evaluation in the risk assessment.

2.1.2 Risk-Based Screening Levels

An RBSL is a chemical concentration in a particular medium that yields a given cancer risk or hazard quotient (HQ) (e.g., 10^{-7} cancer risk; 0.1 HQ) under a given set of conditions. For Wainwright, the RBSLs were calculated for soil based on EPA default reasonable maximum exposure (RME) parameters (EPA 1991a). In developing the RBSLs, the most recent toxicity factors available from the Integrated Risk Information System (IRIS) and the Health Effects Assessment Summary Tables (HEAST) were used. IRIS and HEAST are databases of toxicity information for human health risk assessment maintained by the Environmental Criterion Assessment Office (ECAO) of the EPA. The information presented on IRIS represents the agency's consensus regarding the toxicity of chemicals released to the environment. Toxicity factors that EPA has withdrawn from IRIS and HEAST, or available from other sources were not used in this risk assessment.

2.1.2.1 Formulae for Calculating RBSLs. The RBSL concentrations were derived using EPA Region 10 guidance (1991a). The equations presented by EPA (1991a) are also presented in a slightly different form in the Risk Assessment Guidance for Superfund Volume I, Part B (EPA 1991b). Exposure assessment and risk characterization algorithms for human health risk assessments use site-specific contaminant concentration data, factors describing exposure, and toxicity dose-response values (e.g., reference doses or carcinogen slope factors). These risk assessment algorithms were solved for the concentration term to derive the RBSL for soil and ground or surface water. The algorithms are summarized as follows:

$$\text{Risk} = C \times \left(\frac{CR \times EFD}{BW \times AT}\right) \times SF \quad \text{or} \quad HQ = C \times \left(\frac{CR \times EFD}{BW \times AT}\right) / RfD \qquad \textbf{EQUATION 1, 2}$$

Risk = Target Cancer Risk
C = Concentration
CR = Contact Rate
EFD = Exposure Frequency and Duration
BW = Body Weight
AT = Averaging Time
SF = Slope Factor
HQ = Target Hazard Quotient
RfD = Reference Dose

RBSLs are calculated based on a specific target cancer risk or HQ. EPA (1991a) recommends that a 1×10^{-7} target cancer risk and a target noncancer HQ of 0.1 be used for soil and a 1×10^{-6} risk and 0.1 HQ be used for surface water. The lower target cancer risk is used for screening soil because additional pathways, such as dermal contact and inhalation, are not accounted for by the calculations (EPA 1991a).

Equations (1) and (2) shown above are rearranged to solve for the concentration term (i.e., the RBSL):

$$C = \text{Risk} / \left(\left(\frac{CR \times EFD}{BW \times AT}\right) \times SF \right) \quad \text{or} \quad C = HQ / \left(\left(\frac{CR \times EFD}{BW \times AT}\right) / RfD \right) \qquad \textbf{EQUATION 3, 4}$$

Surface Water Ingestion Equations. Using standard default exposure factors (EPA 1989b) for water ingestion, the equation for cancer risk from drinking water ingestion becomes:

$$\text{Risk} = C\ (\mu g/L) \times 0.001\ mg/\mu g \times \left(\frac{2\ L/day \times 350\ day/year \times 30\ year}{70\ kg \times 70\ year \times 365\ day/year}\right) \times SF_o \qquad \textbf{EQUATION 5}$$

Equation 5 can be rearranged to solve for an RBSL with, for example, a target cancer risk of 10^{-6}:

$$C\ (\mu g/L) = 10^{-6} \times 1{,}000\ \mu g/mg \Big/ \left[\left(\frac{2\ L/day \times 350\ day/year \times 30\ year}{70\ kg \times 70\ year \times 365\ day/year}\right) \times SF_o\right] \qquad \textbf{EQUATION 6}$$

For non-carcinogens, the equation for the HQ for drinking water ingestion is:

$$HQ = C\ (\mu g/L) \times 0.001\ mg/\mu g \times \left(\frac{2\ L/day \times 350\ day/year \times 30\ year}{70\ kg \times 30\ year \times 365\ day/year}\right) / RfD_o \qquad \textbf{EQUATION 7}$$

Equation 7 can be rearranged to provide an equation for the concentration that represents an HQ of 1 from ingestion:

$$C\ (\mu g/L) = 1 \times 1{,}000\ \mu g/mg \Big/ \left[\left(\frac{2\ L/day \times 350\ day/year \times 30\ year}{70\ kg \times 30\ year \times 365\ day/year}\right) / RfD_o\right] \qquad \textbf{EQUATION 8}$$

Soil or Sediment Ingestion Equations. The equation for calculating carcinogenic risk from soil or sediment ingestion, combining child and adult exposure, is as follows:

$$\textbf{EQUATION 9}$$

$$\text{Risk} = C\ (mg/kg) \times 0.000001\ kg/mg \times$$

$$\left[\left(\left(\frac{200_c\ mg/day \times 350_c\ day/year \times 6\ year}{15_c\ kg \times 365\ day/year}\right) + \left(\frac{100_a\ mg/day \times 350_a\ day/year \times 24\ year}{70_a\ kg \times 365\ day/year}\right)\right) / 70\ year\right] \times SF_o$$

Equation 9 can be rearranged to solve for the concentration that represents a target cancer risk of 10^{-7}:

$$\textbf{EQUATION 10}$$

$$C\ (mg/kg) = 10^{-7} \times 1{,}000{,}000\ kg/mg/$$

$$\left[\left(\left(\frac{200_c\ mg/day \times 350_c\ day/year \times 6\ year}{15_c\ kg \times 365\ day/year}\right) + \left(\frac{100_a\ mg/day \times 350_a\ day/year \times 24\ year}{70_a\ kg \times 365\ day/year}\right)\right) / 70\ year\right] \times SF_o$$

For non-carcinogens in soil, Equation 11 is used to calculate the HQ:

$$HQ = C\ (mg/kg) \times 0.000001\ kg/mg \times \left[\left(\frac{200_c\ mg/day \times 350_c\ day/year \times 6\ year}{15_c\ kg \times 365\ day/year}\right) + \left(\frac{100_a\ mg/day \times 350_a\ day/year \times 24\ year}{70_a\ kg \times 365\ day/year}\right) / 30\ year\right] / RfD_o \qquad \textbf{EQUATION 11}$$

Equation 11 can be rearranged to solve for the concentration that represents an HQ of 0.1:

$$C\ (mg/kg) = 0.1 \times 1{,}000{,}000\ mg/kg\ / \left[\left(\left(\left(\frac{200_c\ mg/day \times 350_x\ day/year \times 6\ year}{15_c\ kg \times 365\ day/year}\right) + \left(\frac{100_a\ mg/day \times 350_a\ day/year \times 24\ year}{70_a\ kg \times 365\ day/year}\right)\right) / 30\ year\right) / RfD_o\right] \qquad \textbf{EQUATION 12}$$

2.1.3 Screening of Chemicals by Comparing Maximum Detected Concentrations of Essential Human Nutrients

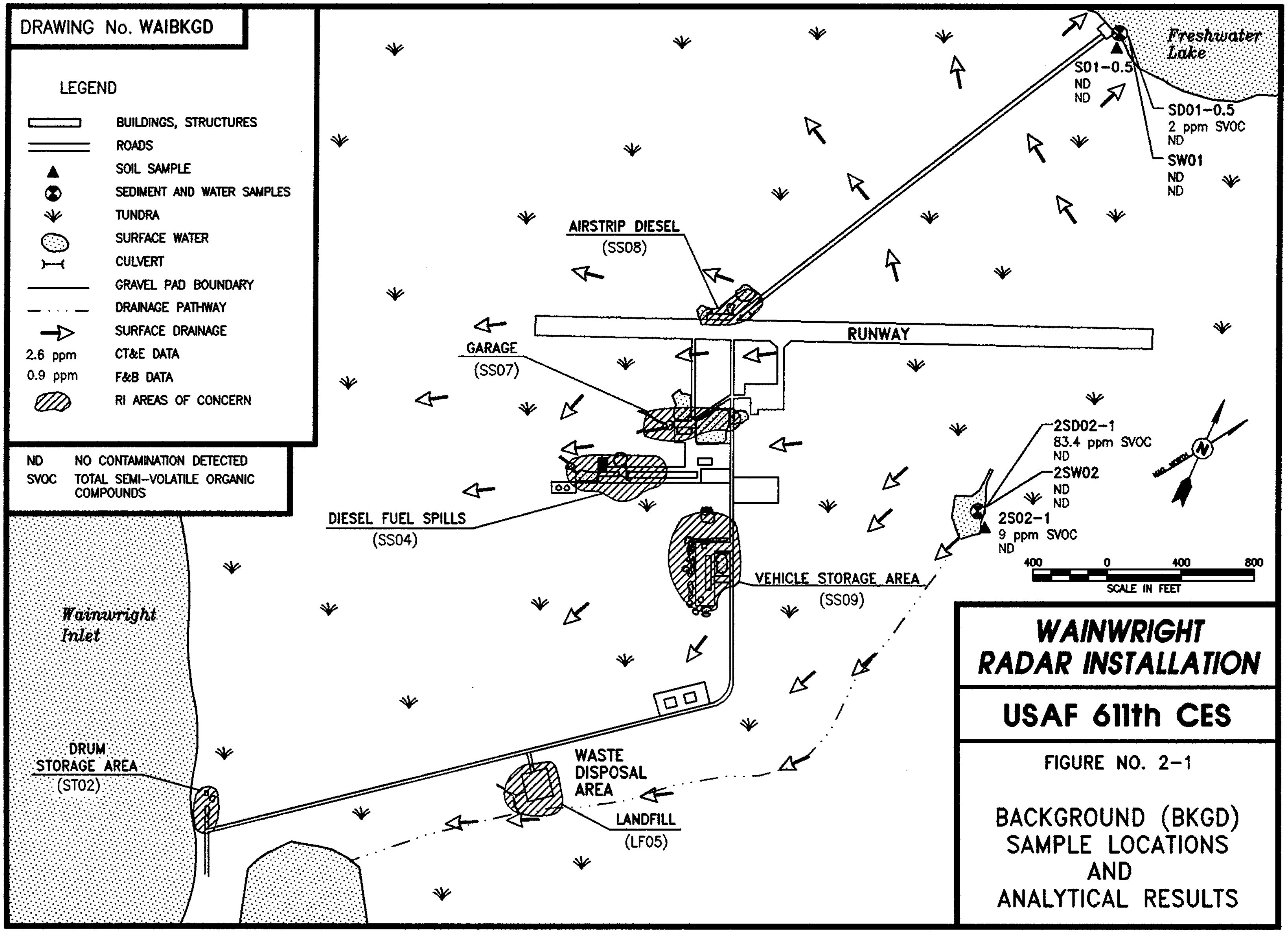
Based on EPA's guidance (1991a), calcium, magnesium, potassium, iron, and sodium are considered to be essential human nutrients and were eliminated from the human health risk assessment at the screening stage. These chemicals are often detected but are not toxic to humans except at extremely high doses. No quantitative toxicity information is available for these elements from EPA sources; therefore, these metals are not selected as COCs for this risk assessment.

2.1.4 Concentrations of Organic and Inorganic Constituents in Background Samples

Seven samples were collected upgradient of the radar installation to determine the concentrations of naturally occurring organic and inorganic constituents in soil, sediment, and surface water (Figure 2-1). Soil and sediment background samples were collected at a depth of zero to six inches. Although some naturally occurring compounds were detected in some of the soil, sediment, and surface water background samples in the diesel range petroleum hydrocarbons (DRPH) analyses, the organic concentration in background samples is assumed to be non-detect. This conservative approach was used because it is not possible to determine to what degree, if any, the DRPH detected in site samples were naturally occurring compounds.

In order to obtain a representative range of background inorganic (metal) concentrations in soil, sediment and surface water of the North Slope, 44 samples (29 soil or sediment, and 15 water) from seven North Slope radar installations were collected. The seven installations are Barter Island, Bullen Point, Oliktok Point, Point Lonely, Point Barrow, Point Lay, and Wainwright. Approximately five soil or sediment and two surface water background samples were collected and analyzed for metals at each of the seven radar installations. Analytical results for background samples collected at the Wainwright installation are presented in Appendix G.

THIS PAGE INTENTIONALLY LEFT BLANK



WAINWRIGHT RADAR INSTALLATION

USAF 611th CES

FIGURE NO. 2-1

BACKGROUND (BKGD) SAMPLE LOCATIONS AND ANALYTICAL RESULTS

THIS PAGE INTENTIONALLY LEFT BLANK

2.1.5 Selection of Chemicals of Concern

Soil and Sediment. The maximum concentrations of the chemicals detected in soil or sediment samples at the Wainwright installation and not considered to be essential human nutrients were compared, on a site-by-site basis, to the corresponding background concentrations, RBSLs, and where available, federal or state ARARs. Chemicals detected without an RBSL or ARAR were retained as COCs if concentrations exceeded background levels. A chemical with an RBSL or ARAR was selected as a COC for soil and sediment if the maximum concentration at which the chemical was detected exceeded the corresponding background concentration and the RBSL (based either on cancer risk or noncancer hazard) or ARAR (Table 2-1). Thus, for example, the maximum concentration of DRPH at the Diesel Fuel Spills (SS04), 4,900 mg/kg, exceeds the background range and the state ARAR of 500 mg/kg. Therefore, DRPH was selected as a COC for the soils at the Diesel Fuel Spills site.

The COCs for soil/sediment at each site were compared to background concentrations, RBSLs, and ARARs in Table 2-1. The chemicals retained as COCs exceed background concentrations and the RBSL, or an ARAR. A chemical was not retained if the level detected was less than the corresponding RBSL and ARAR, even though background levels were exceeded. The COCs selected that do not have an RBSL or an ARAR are discussed below. The COCs selected at each site that exceed an RBSL, ARAR, or both, are discussed in Sections 2.1.5.1 to 2.1.5.6.

Surface Water. The maximum concentrations of the chemicals detected in surface water samples at Wainwright were compared, on a site-by-site basis, to the corresponding background concentrations, RBSLs, and where available, federal or state ARARs. Chemicals detected without an RBSL or ARAR were retained as COCs if concentrations exceeded background levels. A chemical with an RBSL or ARAR was selected as a COC for surface water if the maximum concentration at which the chemical was detected exceeded the corresponding background concentration, and the RBSL (based either on cancer risk or noncancer hazard) or ARAR (Table 2-1). Thus, for example, the maximum concentration of 1,2-dichloroethane at the Landfill (LF05), 6.2 μg/L, exceeds the background concentration of <1 μg/L (not detected) and the RBSL based on cancer risk of 0.934 μg/L. Therefore, 1,2-dichloroethane was selected as a COC for the surface water at the Landfill (LF05) site.

The maximum concentration of metals in surface water was normally detected in the total metal analyses. The dissolved metal concentration was used in the risk assessment if the value reported exceeded the total metal concentration. Metals concentrations in the risk evaluation are total metals unless specifically noted as dissolved.

The COCs for surface water at each site were compared to background concentrations, RBSLs, and ARARs in Table 2-1. The chemicals retained as COCs exceed background concentrations, the RBSL, or an ARAR. A chemical was not retained if the level detected was less than the corresponding RBSL and ARAR, even through background levels were exceeded. The COCs selected that exceed background levels, but do not have an RBSL or ARAR are discussed below.

TABLE 2-1. IDENTIFICATION OF CONTAMINANTS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED AT WAINWRIGHT

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL[1] CANCER	RBSL[1] NON-CANCER	ARAR[2]	CHEMICAL OF CONCERN
Drum Storage Area (ST02)	Soil	Aluminum	9,600	mg/kg	1,500-25,000	--	--	--	No
		Barium	180	mg/kg	27-390	--	1,890	--	No
		Calcium	15,000	mg/kg	360-59,000	--	--	--	No
		Cobalt	15	mg/kg	<5.1-12	--	--	--	**Yes***
		Copper	9.3	mg/kg	<2.7-45	--	999	--	No
		Iron	110,000	mg/kg	5,400-35,000	--	--	--	No
		Lead	19	mg/kg	<5.1-22	--	--	500[a]	No
		Magnesium	5,300	mg/kg	360-7,400	--	--	--	No
		Manganese	1,400	mg/kg	25-290	--	3,780	--	No
		Nickel	24	mg/kg	4.2-46	--	540	--	No
		Potassium	930	mg/kg	<300-2,200	--	--	--	No
		Sodium	840	mg/kg	<160-680	--	--	--	No
		Vanadium	39	mg/kg	6.3-59	--	189	--	No
		Zinc	66	mg/kg	9.2-95	--	8,100	--	No
Diesel Fuel Spills (SS04)	Soil/Sediment	DRPH	4,900J	mg/kg	<50-<300J	--	--	500[b]	**Yes**
		GRPH	120NJ	mg/kg	<2J-<5J	--	--	100[b]	**Yes**
		Ethylbenzene	7NJ	mg/kg	<0.020-<0.400	--	2,700	--	No
		p-Isopropyltoluene	0.237	mg/kg	<0.020-<0.400	--	5,400	--	No
		Naphthalene	0.851	mg/kg	<0.020-<32.0	--	1,100	--	No
		1,2,4-Trimethylbenzene	14.4	mg/kg	<0.020-<0.400	--	--	--	**Yes***
		1,3,5-Trimethylbenzene	5.49	mg/kg	<0.020-<0.400	--	--	--	**Yes***
		Xylenes	17NJ	mg/kg	<0.040-<0.200	--	54,000	--	No
Landfill (LF05)	Soil/Sediment	DRPH	60	mg/kg	<50-<300J	--	--	500[b]	No
		GRPH	200J	mg/kg	<2J-<5J	--	--	100[b]	**Yes**

TABLE 2-1. IDENTIFICATION OF CONTAMINANTS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED (CONTINUED)

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL[1] CANCER	RBSL[1] NON-CANCER	ARAR[2]	CHEMICAL OF CONCERN
Landfill	Soil/Sediment	di-n-Butylphthalate	37.6J	mg/kg	1.69U-83.4J	--	2,700	8,000[c]	No
(LF05)	(Continued)	Ethylbenzene	1.5J	mg/kg	<0.020-<0.400	--	2,700		No
(Continued)		Toluene	0.205	mg/kg	<0.020-<0.400	--	5,400	--	No
		1,3,5-Trimethylbenzene	0.247	mg/kg	<0.020-<0.400	--	--	--	Yes*
		Xylenes	8J	mg/kg	<0.040-<0.200	--	54,000	--	No
		Aluminum	15,000	mg/kg	1,500-25,000	--	--	--	No
		Barium	420	mg/kg	27-390	--	1,890	--	No
		Cadmium	72	mg/kg	<3.0-<36	--	27	--	Yes
		Calcium	4,950	mg/kg	360-59,000	--	--	--	No
		Chromium	26	mg/kg	<4.3-47	--	135	--	No
		Cobalt	12	mg/kg	<5.1-12	--	--	--	No
		Copper	41	mg/kg	<2.7-45	--	999	--	No
		Iron	99,000	mg/kg	5,400-35,000	--	--	--	No
		Lead	37	mg/kg	<5.1-22	--	--	500[a]	No
		Magnesium	3,100	mg/kg	360-7,400	--	--	--	No
		Manganese	1,100J	mg/kg	25-290	--	3,780	--	No
		Nickel	25	mg/kg	4.2-46	--	540	--	No
		Potassium	1,500J	mg/kg	<300-2,200	--	--	--	No
		Sodium	1,500	mg/kg	<160-680	--	--	--	No
		Vanadium	43	mg/kg	6.3-59	--	189	--	No
		Zinc	150	mg/kg	9.2-95	--	8,100	--	No
	Surface Water	1,2-Dichloroethane	6.2	µg/L	<1	0.934	--	5[d]	Yes
		Aluminum	2,100	µg/L	<100-350	--	--	--	Yes*
		Barium	230	µg/L	<50-93	--	256	2,000[e]	No
		Calcium	24,000	µg/L	4,500-88,000	--	--	--	No

TABLE 2-1. IDENTIFICATION OF CONTAMINANTS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED (CONTINUED)

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL[1]		ARAR[2]	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Landfill	Surface Water	Iron	23,000	µg/L	100-2,800	--	--	--	No
(LF05)	(Continued)	Magnesium	26,000	µg/L	<5,000-53,000	--	--	--	No
(Continued)		Manganese	150	µg/L	<50-510	--	18.3	--	No
		Potassium	9,500	µg/L	<5,000	--	--	--	No
		Sodium	110,000	µg/L	8,400-410,000	--	--	--	No
		Zinc	230J	µg/L	<50-160	--	1,100	5,000[f]	No
Garage	Soil/Sediment	DRPH	120,000J	mg/kg	<50-<300J	--	--	500[b]	**Yes**
(SS07)		GRPH	120J	mg/kg	<2J-<5J	--	--	100[b]	**Yes**
		RRPH	77,000	mg/kg	<100-<600	--	--	2,000[b]	**Yes**
		Ethylbenzene	4NJ	mg/kg	<0.020-<0.400	--	2,700	--	No
		p-Isopropyltoluene	0.502	mg/kg	<0.02-<0.4	--	--	--	**Yes***
		Naphthalene	0.393	mg/kg	<0.020-<32.0	--	1,100	--	No
		Tetrachloroethene	11.5	mg/kg	<0.020-<0.400	1.23	270	--	**Yes**
		1,2,4-Trimethylbenzene	0.714	mg/kg	<0.020-<0.400	--	--	--	**Yes***
		1,3,5-Trimethylbenzene	5.36	mg/kg	<0.02-<0.4	--	--	--	**Yes***
		Xylenes	18NJ	mg/kg	<0.040-<0.200	--	54,000	--	No
		Aluminum	2,800	mg/kg	1,500-25,000	--	--	--	No
		Barium	240	mg/kg	27-390	--	1,890	--	No
		Calcium	6,100	mg/kg	360-59,000	--	--	--	No
		Chromium	30	mg/kg	<4.3-47	--	135	--	No
		Cobalt	8.6	mg/kg	<5.1-12	--	--	--	No
		Copper	39	mg/kg	<2.7-45	--	999	--	No
		Iron	114,000	mg/kg	5,400-35,000	--	--	--	No
		Lead	130	mg/kg	<5.1-22	--	--	500[a]	No
		Magnesium	3,300	mg/kg	360-7,400	--	--	--	No

TABLE 2-1. IDENTIFICATION OF CONTAMINANTS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED (CONTINUED)

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL[1]		ARAR[2]	CHEMICAL OF CONCERN
						CANCER	NON-CANCER		
Garage (SS07) (Continued)	Soil/Sediment (Continued)	Manganese	1,250	mg/kg	25-290	--	3,780	--	No
		Nickel	29	mg/kg	4.2-46	--	540	--	No
		Potassium	425	mg/kg	<300-2,200	--	--	--	No
		Sodium	100	mg/kg	<160-680	--	--	--	No
		Vanadium	34	mg/kg	6.3-59	--	189	--	No
		Zinc	240	mg/kg	9.2-95	--	8,100	--	No
	Surface Water	1,2-Dichloroethane	1.8J	µg/L	<1	0.934	--	5[d]	**Yes**
		bis-(2Ethylhexyl)Phthalate	16	µg/L	<10-<13	6.07	73	--	**Yes**
		Barium	240	µg/L	<50-93	--	256	2,000[e]	No
		Calcium	37,000	µg/L	4,500-8,800	--	--	--	No
		Iron	2,900	µg/L	180-2,800	--	--	--	No
		Magnesium	62,000	µg/L	<5,000-53,000	--	--	--	No
		Sodium	51,000	µg/L	8,400-410,000	--	--	--	No
		Zinc	440	µg/L	<50-160	--	1,100	5,000[f]	No
Airstrip Diesel (SS08)	All sediment and surface water samples are non-detect at this site.								
Vehicle Storage Area (SS09)	Soil/Sediment	Naphthalene	0.072	mg/kg	<0.020-<32.0	--	1,110	--	No
		Benzyl alcohol	0.694	mg/kg	<0.20-<32.0	--	8,100	--	No
		Tetrachloroethene	0.330	mg/kg	<0.020-<0.400	1.23	270	--	No
		Toluene	0.172	mg/kg	<0.020-<0.400	--	5,400	--	No
		Trichloroethene	0.062	mg/kg	<0.020-<0.400	5.8	--	--	No
		1,2,4-Trimethylbenzene	0.042	mg/kg	<0.020-<0.400	--	--	--	**Yes***
		Xylenes	0.125	mg/kg	<0.040-<0.800	--	54,000	--	No
		Aluminum	2,800	mg/kg	1,500-25,000	--	--	--	No
		Barium	170	mg/kg	27-390	--	1,890	--	No
		Calcium	3,800	mg/kg	360-59,000	--	--	--	No

TABLE 2-1. IDENTIFICATION OF CONTAMINANTS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED (CONTINUED)

SITE	MATRIX	CHEMICAL DETECTED	MAXIMUM CONCENTRATION	UNITS	BACKGROUND RANGE	RBSL[1] CANCER	RBSL[1] NON-CANCER	ARAR[2]	CHEMICAL OF CONCERN
Vehicle Storage Area	Soil/Sediment	Chromium	11	mg/kg	<4.3-47	--	135	--	No
(SS09)	(Continued)	Cobalt	3.8	mg/kg	<5.1-12	--	--	--	No
(Continued)		Copper	9.4	mg/kg	<2.7-45	--	999	--	No
		Iron	24,400	mg/kg	5,400-35,000	--	--	--	No
		Magnesium	1,800	mg/kg	360-7,400	--	--	--	No
		Manganese	230	mg/kg	25-290	--	3,780	--	No
		Nickel	11	mg/kg	4.2-46	--	540	--	No
		Potassium	690J	mg/kg	<300-2,200	--	--	--	No
		Sodium	290J	mg/kg	<160-680	--	--	--	No
		Vanadium	17	mg/kg	6.3-59	--	189	--	No
		Zinc	32	mg/kg	9.2-95	--	8,100	--	No
	Surface Water	1,2-Dichloroethane	1.6	µg/L	<1	0.934	--	5[d]	**Yes**
		Aluminum	9,700	µg/L	<100-350	--	--	--	**Yes***
		Barium	750	µg/L	<50-93	--	256	2,000[e]	**Yes**
		Calcium	71,000	µg/L	4,500-88,000	--	--	--	No
		Iron	130,000	µg/L	180-2,800	--	--	--	No
		Magnesium	57,000	µg/L	<5,000-53,000	--	--	--	No
		Manganese	3,800	µg/L	<50-510	--	18.3	--	**Yes**
		Nickel	51	µg/L	<50	--	73	100[g]	No
		Sodium	45,000	µg/L	8,400-410,000	--	--	--	No
		Vanadium	63	µg/L	<50	--	25.6	--	**Yes**
		Zinc	3,300	µg/L	<50-160	--	1,100	--	**Yes**

* Chemicals without an RBSL or ARAR are considered chemicals of potential concern and are discussed in Section 2.1.5.
1 R[illegible]k-Based Screening Levels.

TABLE 2-1. IDENTIFICATION OF CONTAMINANTS OF CONCERN: COMPARISON OF MAXIMUM CONCENTRATIONS TO RISK-BASED SCREENING LEVELS, ARARS, AND BACKGROUND EVALUATION OF CHEMICALS DETECTED (CONTINUED)

2 Applicable or Relevant and Appropriate Requirements.
J Result is an estimate.
N The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".
U Compound is not present above the concentration listed.
a EPA 1991c.
b Target cleanup levels for DRPH, GRPH, and RRPH in soil are based on Alaska Department of Environmental Conservation (ADEC) Non-Underground Storage Tank (UST) guidance and do not necessarily correspond to final site specific cleanup goals.
c 55 FR 30798, Proposed Rule RCRA Corrective Action for SWMUs 40 CFR [Section 264.521 (a)(2)(i-iv)], Health-Based Criteria for Systemic Toxicant.
d MCL, 52 FR 25690 (08 Jul 89).
e MCL, 56 FR 30266 (01 Jul 91).
f Secondary MCL for Zinc, 54 FR 22062 (22 May 89).
g MCL, 57 FR 31776 (17 Jul 92).

Risk Characterization of Chemicals without RBSLs and ARARs. Several chemicals detected above background levels could not be thoroughly screened because an RBSL could not be calculated and no ARAR was available (Table 2-1). A list of these chemicals is presented in Table 2-2. The cancer risk and noncancer hazard for these chemicals cannot, therefore, be quantified. This section is a qualitative discussion of the potential for these chemicals to cause toxicity among the receptor groups identified at the Wainwright installation. The essential human nutrients were discussed in Section 2.1.3 and will not be discussed further here. Essential nutrients are not considered COCs in this risk assessment.

The American Petroleum Institute (API) recently published an evaluation of the environmental fate, transport, and toxicity of 12 organic chemicals found frequently in petroleum products. The 12 were selected from a large list of "candidates" based on:

- abundance in crude and refined petroleum products, including residual and used oils;
- chemical/physical properties that represent a range of mobilities in soil and solubilities in aqueous environments; and
- toxicity in mammals and aquatic organisms (API 1994).

One of the chemicals detected at the Wainwright installation, 1,2,4-trimethylbenzene, was selected from the list of 12 chemicals (API 1994) and used in this risk assessment as a surrogate for the chemicals without RBSLs and ARARs. The chemical has a similar chemical structure to, and therefore will represent, the substituted benzenes listed in Table 2-2 that do not have toxicity criteria.

1,2,4-Trimethylbenzene has a low order of toxicity in mammals (API 1994). No effect was observed on the kidneys of rats that received 0.5 or 2.0 g/kg orally five days per week for four weeks. Inhalation of high concentrations of 1,2,4-trimethylbenzene produces central nervous system depression in humans and rats. Lung toxicity, including bronchitis, pneumonitis, and edema, was also observed in humans. 1,2,4-Trimethylbenzene has not been observed to be carcinogenic or mutagenic in laboratory studies of rats and cultured mammalian cells. Potential exposure of receptors to 1,2,4-trimethylbenzene at the Wainwright installation would probably be limited to oral ingestion of soil and at the maximum concentration measured (14.4 mg/kg soil) would be expected to be nontoxic.[1] For the purposes of this risk assessment, 1,2,4-trimethylbenzene is considered to be a reasonable surrogate for the substituted benzenes observed at the Wainwright installation.

[1] Based on the following calculation: assume average daily soil ingestion rate of 200 mg of soil per day and 14.4 mg of 1,2,4-trimethylbenzene per kg of soil (maximum concentration measured at Wainwright installation). This yields a dose of 0.00004 mg of 1,2,4-trimethylbenzene per kg body weight per day. The oral dose of 1,2,4-trimethylbenzene received by rats that showed no kidney effects was equivalent to 2,000 mg of 1,2,4-trimethylbenzene per kg body weight, which is more than 50,000,000 times greater than the estimated dose for potential receptors at the Wainwright installation.

TABLE 2-2. CHEMICALS WITHOUT RBSLS AND ARARS OBSERVED IN THE SOIL, SEDIMENT, OR SURFACE WATER AT THE WAINWRIGHT INSTALLATION

SUBSTITUTED BENZENES
1,2,4-Trimethylbenzene
1,3,5-Trimethylbenzene
p-Isopropyltoluene
ESSENTIAL HUMAN NUTRIENTS
Calcium
Iron
Magnesium
Potassium
Sodium
OTHER
Aluminum
Cobalt

The chemicals without RBSLs and ARARs listed in the "Other" category of Table 2-2 are not expected to pose a significant hazard to the receptor groups identified at the Wainwright installation. Aluminum is the most abundant metal in the earth's crust (Lindsay 1979), and the concentrations measured (maximum concentration 7,500 mg/kg) were below the range generally expected in the lithosphere (10,000 to 300,000 mg/kg). Cobalt was observed in only one soil sample at a concentration of 5.7 mg/kg. The common range of cobalt concentrations in the lithosphere is 1 to 40 mg/kg (Lindsay 1979); therefore, it is not likely that any level of toxicity would result from the cobalt detected at Wainwright. It is not considered a COC in this risk assessment.

In conclusion, the organic chemicals discussed above have been marked in Table 2-1 as COCs to indicate that there is some uncertainty in screening out these chemicals. Without toxicity criteria the potential risks of these chemical cannot be quantified. However, based on the information in this section, and the concentrations measured at the sites, these chemicals are not expected to pose a heath risk.

Exposures to Lead. Exposures to lead may cause adverse noncancerous health effects; however, EPA has not developed an RfD for this chemical. Lead concentrations in soil were compared to EPA's final action level for lead in soil of 500 to 1,000 mg/kg. It is estimated that exposure to soil containing 500 mg of lead per kilogram soil would yield blood lead levels below

TABLE 2-3. SUMMARY OF THE CHEMICALS OF CONCERN AT WAINWRIGHT

SITE	CHEMICALS OF CONCERN* SOIL/SEDIMENT	CHEMICALS OF CONCERN* SURFACE WATER
Drum Storage Area (ST02)	**NONE**	**NONE**
Diesel Fuel Spills (SS04)	DRPH GRPH	**NONE**
Landfill (LF05)	GRPH cadmium	1,2-dichloroethane
Garage (SS07)	DRPH GRPH RRPH tetrachloroethene	1,2-dichloroethane bis(2-ethylhexyl)phthalate
Airstrip Diesel (SS08)	**NONE**	**NONE**
Vehicle Storage Area (SS09)	**NONE**	1,2-dichloroethane barium manganese vanadium zinc

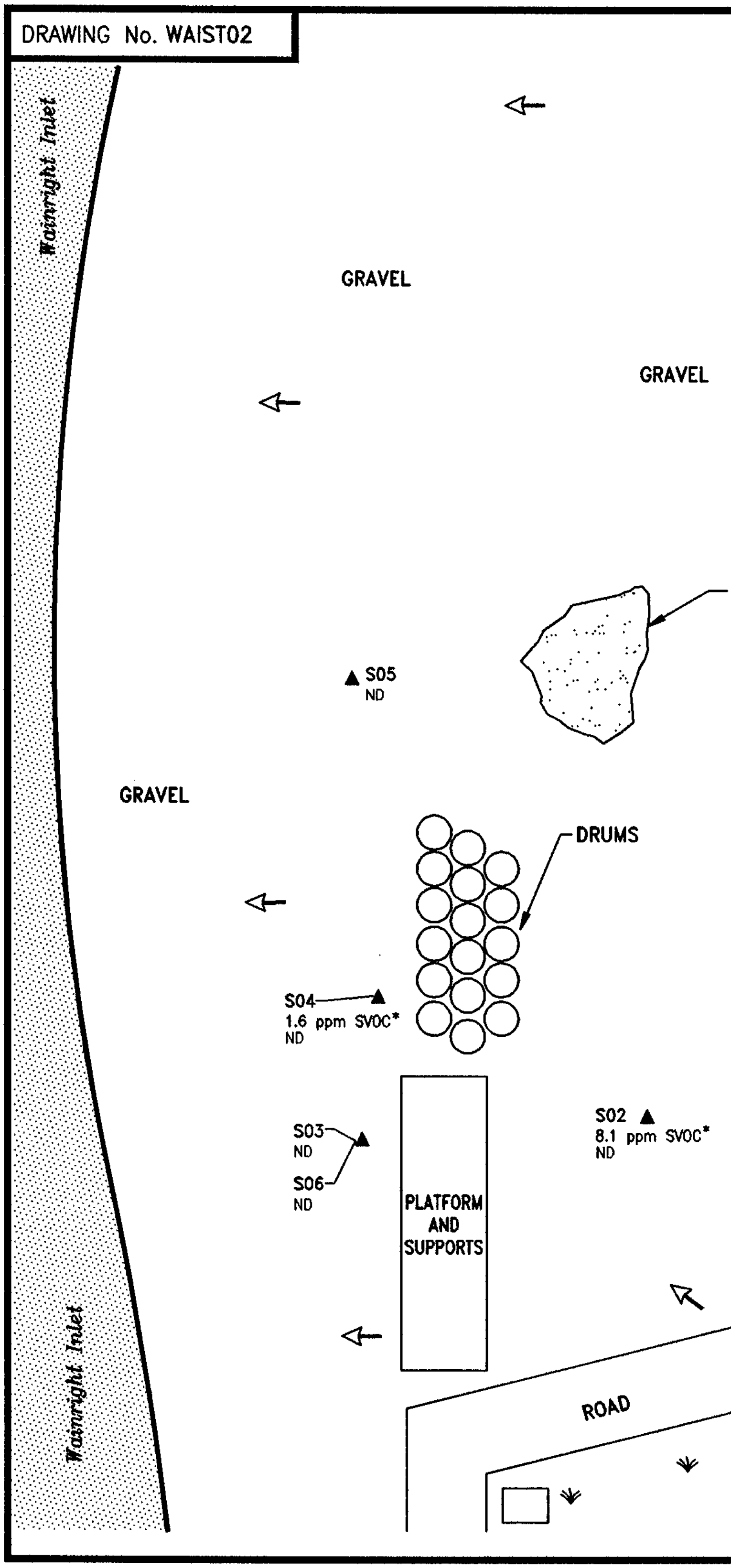
* The summary of COCs on this table includes only those chemicals detected that exceed background levels and an RBSL, ARAR, or both. COCs that exceeded background levels but do not have an RBSL or ARAR are discussed in Section 2.1.5 (Page 2-16).

10 µg/L (a blood lead level of concern) in roughly 99 percent of young children who are not also exposed to excessive lead paint hazards or heavily contaminated soils (EPA 1989c). Lead concentrations did not exceed 500 mg/kg at any sampling location at the Wainwright installation.

Chemicals with RBSLs and/or ARARs. Following are discussions of the COCs at each site that exceeded background levels and an RBSL, ARAR, or both. The site figures presented in Sections 2.1.5.1 through 2.1.5.6 present all organic compounds detected, and only metals detected above background levels at each site. Table 2-3 is a summary of the COCs selected for all of the sites at the Wainwright installation.

2.1.5.1 Drum Storage Area (ST02). No COCs were identified for the soil matrix at the Drum Storage Area (Figure 2-2) based on a comparison of the maximum concentrations of the detected chemicals to their background, RBSL, and ARAR concentrations (Table 2-1).

No surface water bodies were identified at the site; therefore, no surface water COCs were identified.



WAINWRIGHT RADAR INSTALLATION

USAF 611th CES

FIGURE NO. 2-2

DRUM STORAGE AREA (ST02) SAMPLE LOCATIONS AND ANALYTICAL RESULTS

THIS PAGE INTENTIONALLY LEFT BLANK

2.1.5.2 Diesel Fuel Spills (SS04). DRPH and gasoline range petroleum hydrocarbons (GRPH) were identified as COCs for the soil matrix at Diesel Fuel Spills (Figure 2-3). The maximum concentrations of DRPH and GRPH exceeded their background concentrations and the ARAR concentrations for petroleum hydrocarbon contamination of soil (Table 2-1) [Alaska Department of Environmental Conservation (ADEC) 1991].

No COCs were identified for surface water at the site (Figure 2-2) based on a comparison of the maximum concentrations of the detected chemicals to their background, RBSL, or ARAR concentrations (Table 2-1).

2.1.5.3 Landfill (LF05). GRPH and cadmium were identified as COCs for the soil matrix at the Landfill (Figure 2-4). The maximum concentrations of GRPH exceeded the background concentration and the ARAR concentration for petroleum hydrocarbon contamination of soil (Table 2-1) (ADEC 1991). The maximum concentration of cadmium exceeded the background concentration and the noncancer RBSL.

1,2-Dichloroethane was identified as a COC for the surface water at the Landfill (Figure 2-4). The maximum concentration of 1,2-dichloroethane exceeded the carcinogen RBSL and the ARAR for contamination of surface water (Table 2-1).

2.1.5.4 Garage (SS07). DRPH, GRPH, residual range petroleum hydrocarbons (RRPH), and tetrachloroethene were identified as COCs for the soil matrix at the Garage (Figure 2-5). The maximum concentrations of DRPH, GRPH, and RRPH exceeded the ARARs for petroleum hydrocarbon contamination of soil (Table 2-1) (ADEC 1991). The maximum concentration of tetrachloroethene exceeded the carcinogen RBSL for contamination of soil (Table 2-1). Bis(2-ethylhexyl)phthalate and 1,2-dichloroethane were identified as COCs for the surface water at the site (Figure 2-5). The maximum concentrations of bis(2-ethylhexyl)phthalate and 1,2-dichloroethane exceeded the carcinogen RBSLs for contamination of surface water (Table 2-1).

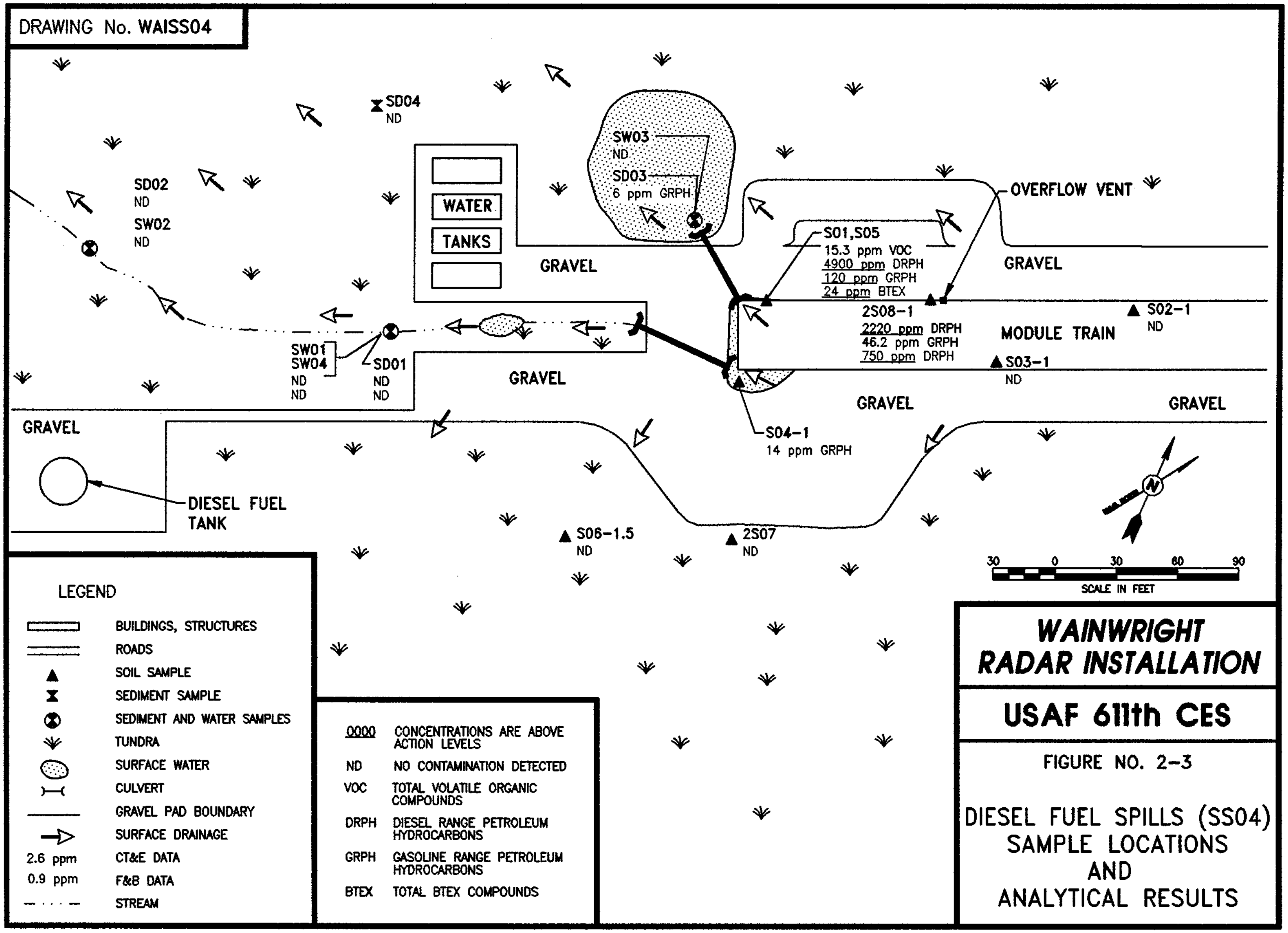
2.1.5.5 Airstrip Diesel (SS08). Five sediment samples and four surface water samples were collected and analyzed for COCs (Figure 2-6). No contamination was detected in any sample. Therefore, no COCs were identified for the Airstrip Diesel site (Table 2-1).

2.1.5.6 Vehicle Storage Area (SS09). No COCs were identified for the soil matrix at the Vehicle Storage Area (Figure 2-7) based on a comparison of the maximum concentrations of the detected chemicals to their background, RBSL, or ARAR concentrations (Table 2-1).

Barium, manganese, vanadium, zinc, and 1,2-dichloroethane were identified as COCs for the surface water at Vehicle Storage Area (Figure 2-7). The maximum concentrations of barium, manganese, vanadium, and zinc exceeded the noncarcinogen RBSLs for contamination of surface water (Table 2-1). The maximum concentration of 1,2-dichloroethane exceeded the carcinogen RBSL for contamination of surface water but was less than the ARAR concentration (Table 2-1).

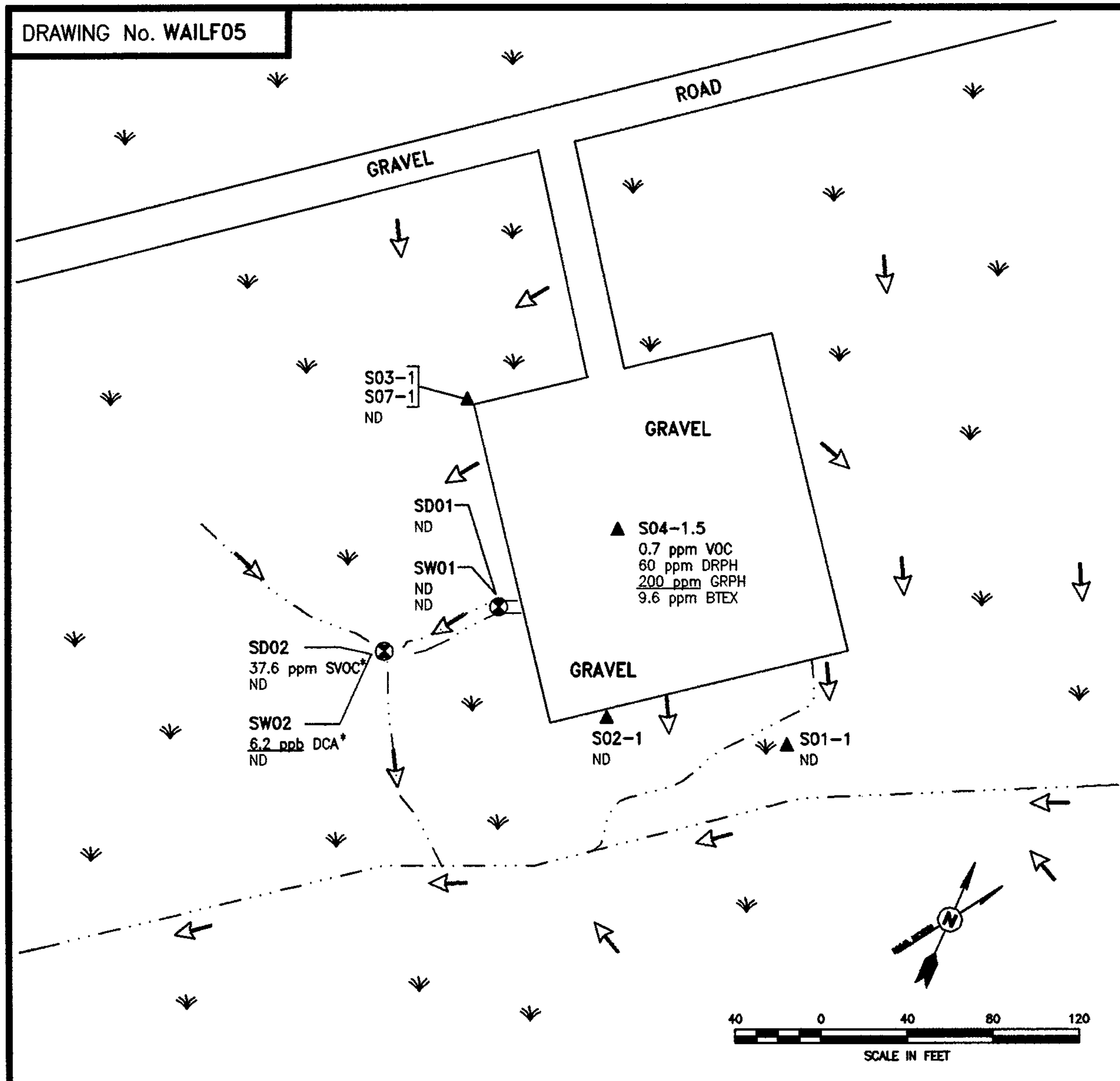
Table 2-3 contains a summary of the COCs identified for each site.

THIS PAGE INTENTIONALLY LEFT BLANK


DRAWING No. WAISS04
SD04
ND
SD02
ND
SW02
ND
WATER
TANKS
GRAVEL
SW03
ND
SD03
6 ppm GRPH
OVERFLOW VENT
S01,S05
15.3 ppm VOC
4900 ppm DRPH
120 ppm GRPH
24 ppm BTEX
2S08-1
2220 ppm DRPH
46.2 ppm GRPH
750 ppm DRPH
MODULE TRAIN
S02-1
ND
S03-1
ND
SW01
SW04
ND
ND
SD01
ND
ND
GRAVEL
GRAVEL
GRAVEL
GRAVEL
GRAVEL
S04-1
14 ppm GRPH
DIESEL FUEL
TANK
S06-1.5
ND
2S07
ND
30 0 30 60 90
SCALE IN FEET
LEGEND
BUILDINGS, STRUCTURES
ROADS
SOIL SAMPLE
SEDIMENT SAMPLE
SEDIMENT AND WATER SAMPLES
TUNDRA
SURFACE WATER
CULVERT
GRAVEL PAD BOUNDARY
SURFACE DRAINAGE
2.6 ppm CT&E DATA
0.9 ppm F&B DATA
STREAM
0000 CONCENTRATIONS ARE ABOVE ACTION LEVELS
ND NO CONTAMINATION DETECTED
VOC TOTAL VOLATILE ORGANIC COMPOUNDS
DRPH DIESEL RANGE PETROLEUM HYDROCARBONS
GRPH GASOLINE RANGE PETROLEUM HYDROCARBONS
BTEX TOTAL BTEX COMPOUNDS
WAINWRIGHT
RADAR INSTALLATION
USAF 611th CES
FIGURE NO. 2-3
DIESEL FUEL SPILLS (SS04)
SAMPLE LOCATIONS
AND
ANALYTICAL RESULTS

THIS PAGE INTENTIONALLY LEFT BLANK

DRAWING No. WAILF05



0000	CONCENTRATIONS ARE ABOVE ACTION LEVELS
ND	NO CONTAMINATION DETECTED
SVOC	TOTAL SEMI-VOLATILE ORGANIC COMPOUNDS
VOC	TOTAL VOLATILE ORGANIC COMPOUNDS
DRPH	DIESEL RANGE PETROLEUM HYDROCARBONS
GRPH	GASOLINE RANGE PETROLEUM HYDROCARBONS
BTEX	TOTAL BTEX COMPOUNDS
DCA	1,2-DICHLOROETHANE

* COMMON LAB CONTAMINANT AND/OR DETECTED IN NUMEROUS BLANK SAMPLES.

LEGEND

	BUILDINGS, STRUCTURES
	ROADS
▲	SOIL SAMPLE
⊗	SEDIMENT AND WATER SAMPLES
	TUNDRA
	GRAVEL PAD BOUNDARY
	DRAINAGE PATHWAY
	SURFACE DRAINAGE
2.6 ppm	CT&E DATA
0.9 ppm	F&B DATA

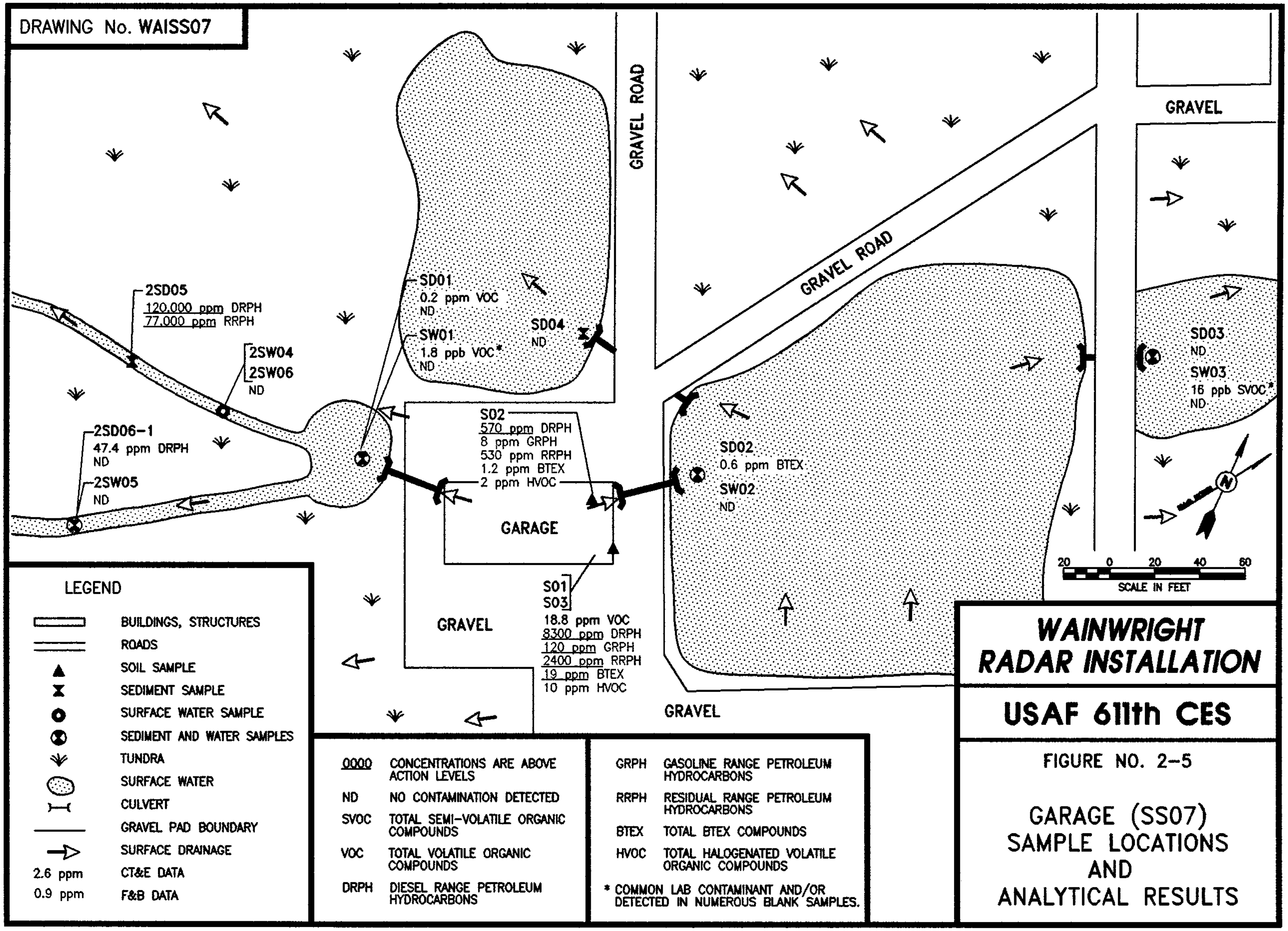
WAINWRIGHT RADAR INSTALLATION

USAF 611th CES

FIGURE NO. 2-4

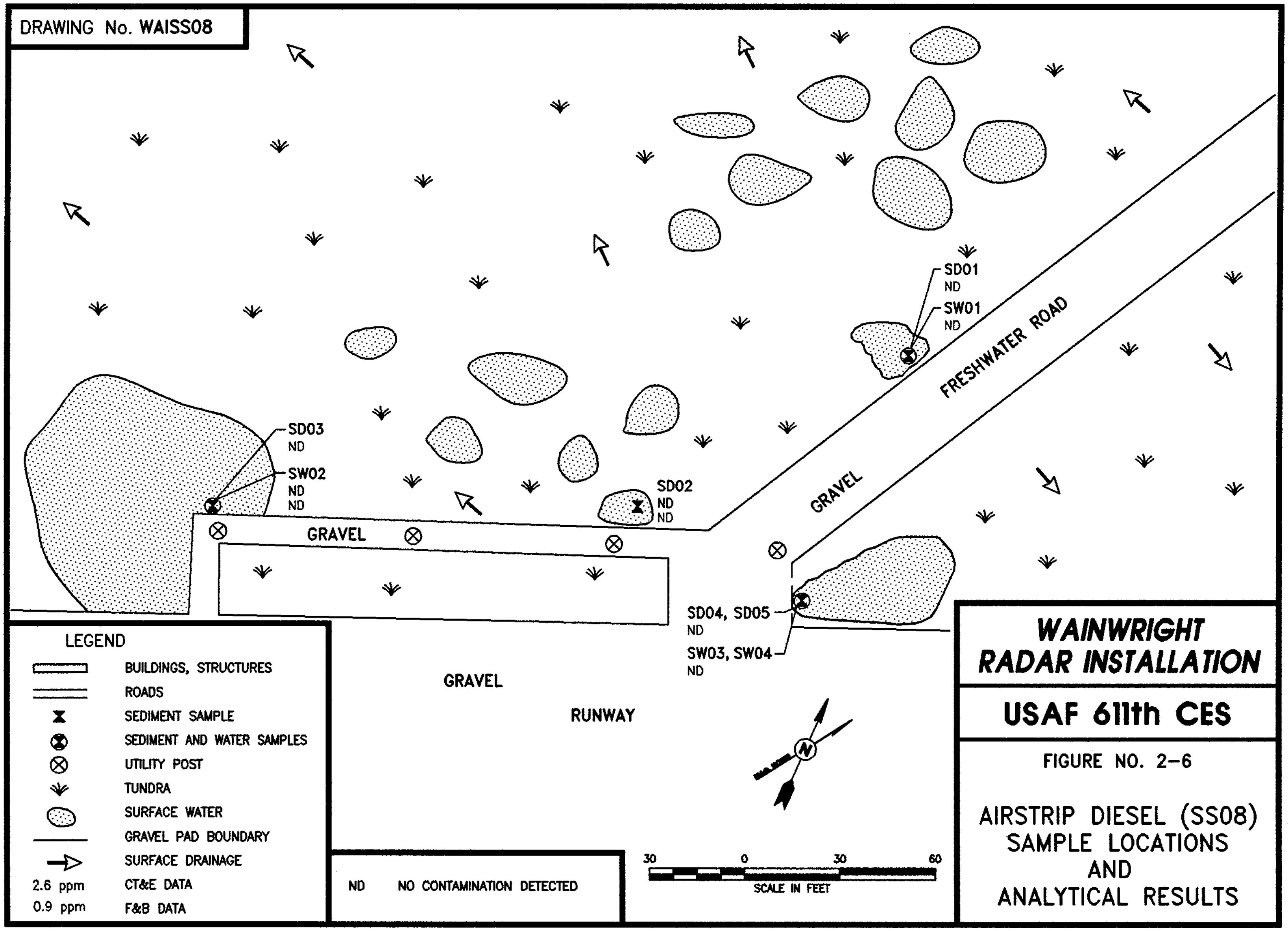
LANDFILL (LF05) SAMPLE LOCATIONS AND ANALYTICAL RESULTS

THIS PAGE INTENTIONALLY LEFT BLANK

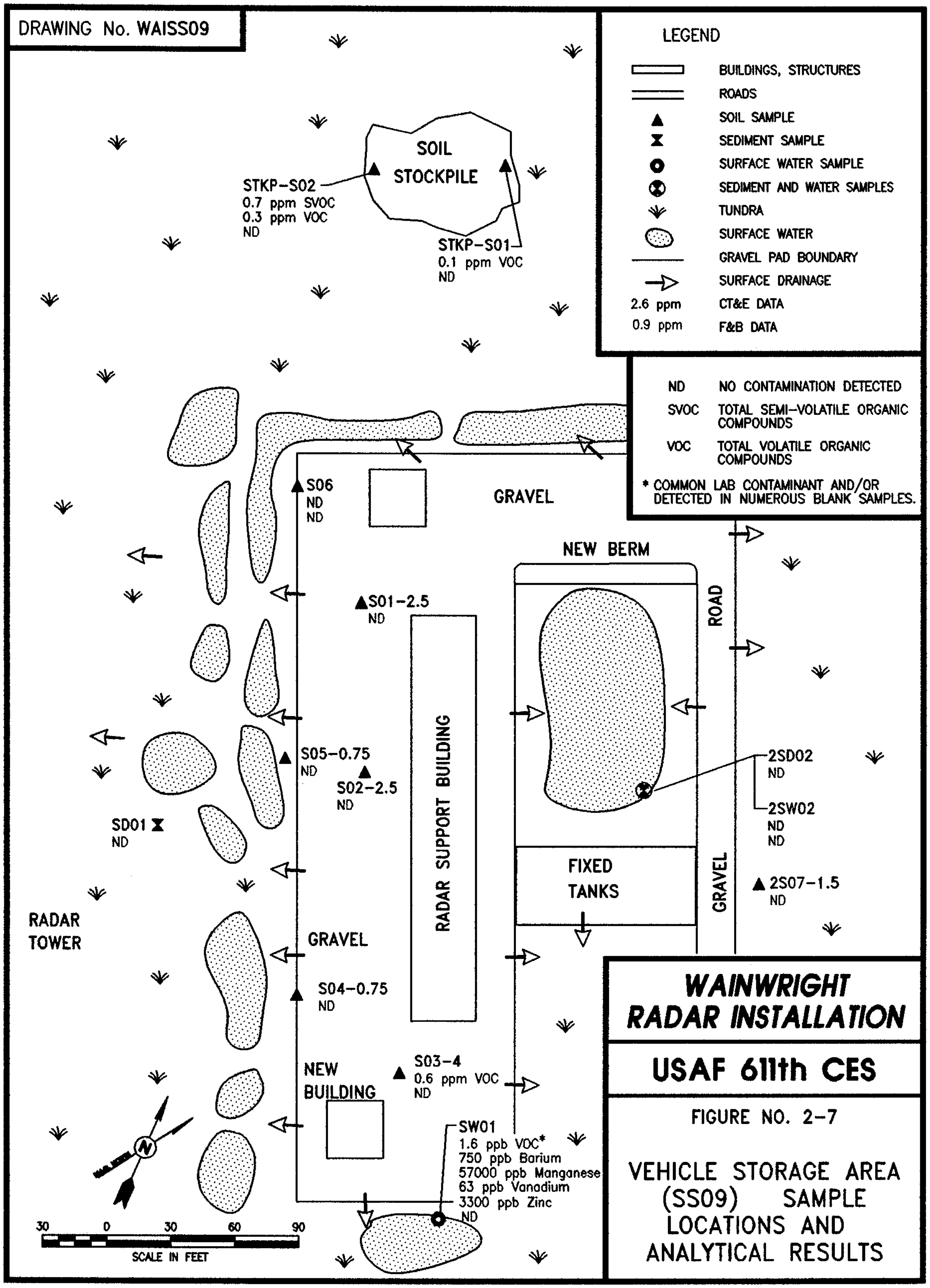


DRAWING No. WAISS07
GRAVEL ROAD
GRAVEL ROAD
GRAVEL
GRAVEL
GRAVEL
GARAGE
2SD05
120,000 ppm DRPH
77,000 ppm RRPH
2SW04
2SW06
ND
2SD06-1
47.4 ppm DRPH
ND
2SW05
ND
SD01
0.2 ppm VOC
ND
SW01
1.8 ppb VOC*
ND
SD04
ND
S02
570 ppm DRPH
8 ppm GRPH
530 ppm RRPH
1.2 ppm BTEX
2 ppm HVOC
S01
S03
18.8 ppm VOC
8300 ppm DRPH
120 ppm GRPH
2400 ppm RRPH
19 ppm BTEX
10 ppm HVOC
SD02
0.6 ppm BTEX
SW02
ND
SD03
ND
SW03
16 ppb SVOC*
ND
N
20 0 20 40 60
SCALE IN FEET
LEGEND
BUILDINGS, STRUCTURES
ROADS
SOIL SAMPLE
SEDIMENT SAMPLE
SURFACE WATER SAMPLE
SEDIMENT AND WATER SAMPLES
TUNDRA
SURFACE WATER
CULVERT
GRAVEL PAD BOUNDARY
SURFACE DRAINAGE
2.6 ppm CT&E DATA
0.9 ppm F&B DATA
0000 CONCENTRATIONS ARE ABOVE ACTION LEVELS
ND NO CONTAMINATION DETECTED
SVOC TOTAL SEMI-VOLATILE ORGANIC COMPOUNDS
VOC TOTAL VOLATILE ORGANIC COMPOUNDS
DRPH DIESEL RANGE PETROLEUM HYDROCARBONS
GRPH GASOLINE RANGE PETROLEUM HYDROCARBONS
RRPH RESIDUAL RANGE PETROLEUM HYDROCARBONS
BTEX TOTAL BTEX COMPOUNDS
HVOC TOTAL HALOGENATED VOLATILE ORGANIC COMPOUNDS
* COMMON LAB CONTAMINANT AND/OR DETECTED IN NUMEROUS BLANK SAMPLES.
WAINWRIGHT RADAR INSTALLATION
USAF 611th CES
FIGURE NO. 2-5
GARAGE (SS07)
SAMPLE LOCATIONS
AND
ANALYTICAL RESULTS

THIS PAGE INTENTIONALLY LEFT BLANK


DRAWING No. WAISS08
SD01
ND
SW01
ND
FRESHWATER ROAD
GRAVEL
SD02
ND
ND
SD03
ND
SW02
ND
ND
GRAVEL
SD04, SD05
ND
SW03, SW04
ND
GRAVEL
RUNWAY
LEGEND
BUILDINGS, STRUCTURES
ROADS
SEDIMENT SAMPLE
SEDIMENT AND WATER SAMPLES
UTILITY POST
TUNDRA
SURFACE WATER
GRAVEL PAD BOUNDARY
SURFACE DRAINAGE
2.6 ppm CT&E DATA
0.9 ppm F&B DATA
ND NO CONTAMINATION DETECTED
30 0 30 60
SCALE IN FEET
N
WAINWRIGHT
RADAR INSTALLATION
USAF 611th CES
FIGURE NO. 2-6
AIRSTRIP DIESEL (SS08)
SAMPLE LOCATIONS
AND
ANALYTICAL RESULTS

THIS PAGE INTENTIONALLY LEFT BLANK


DRAWING No. WAISS09
LEGEND
BUILDINGS, STRUCTURES
ROADS
SOIL SAMPLE
SEDIMENT SAMPLE
SURFACE WATER SAMPLE
SEDIMENT AND WATER SAMPLES
TUNDRA
SURFACE WATER
GRAVEL PAD BOUNDARY
SURFACE DRAINAGE
2.6 ppm CT&E DATA
0.9 ppm F&B DATA
ND NO CONTAMINATION DETECTED
SVOC TOTAL SEMI-VOLATILE ORGANIC COMPOUNDS
VOC TOTAL VOLATILE ORGANIC COMPOUNDS
* COMMON LAB CONTAMINANT AND/OR DETECTED IN NUMEROUS BLANK SAMPLES.
SOIL STOCKPILE
STKP-S02
0.7 ppm SVOC
0.3 ppm VOC
ND
STKP-S01
0.1 ppm VOC
ND
S06
ND
ND
GRAVEL
NEW BERM
ROAD
S01-2.5
ND
RADAR SUPPORT BUILDING
S05-0.75
ND
S02-2.5
ND
2SD02
ND
2SW02
ND
ND
SD01
ND
FIXED TANKS
GRAVEL
2S07-1.5
ND
RADAR TOWER
GRAVEL
S04-0.75
ND
NEW BUILDING
S03-4
0.6 ppm VOC
ND
SW01
1.6 ppb VOC*
750 ppb Barium
57000 ppb Manganese
63 ppb Vanadium
3300 ppb Zinc
ND
N
30 0 30 60 90
SCALE IN FEET
WAINWRIGHT RADAR INSTALLATION
USAF 611th CES
FIGURE NO. 2-7
VEHICLE STORAGE AREA (SS09) SAMPLE LOCATIONS AND ANALYTICAL RESULTS

THIS PAGE INTENTIONALLY LEFT BLANK

2.2 EXPOSURE ASSESSMENT

The exposure assessment section of a baseline human health risk assessment identifies and describes potential receptors and the exposure pathways by which exposure may occur, and estimates the magnitude of those exposures. This section includes an analysis of which pathways are complete (Section 2.2.1), migration and fate of COCs (Section 2.2.2), an estimation of the total intake of the chemicals (Section 2.2.3), and a summary of how the average daily dose (ADD) was calculated (Section 2.2.4).

2.2.1 Pathway Analysis

Pathway analysis involves the evaluation of the components of potential exposure pathways and a determination of whether each pathway is complete. An exposure pathway describes the course a chemical will take from a source to an exposure point where a receptor can come into contact with the chemical. A complete exposure pathway has five components:

- source of contamination;
- release mechanism;
- transport mechanism;
- exposure point; and
- receptor.

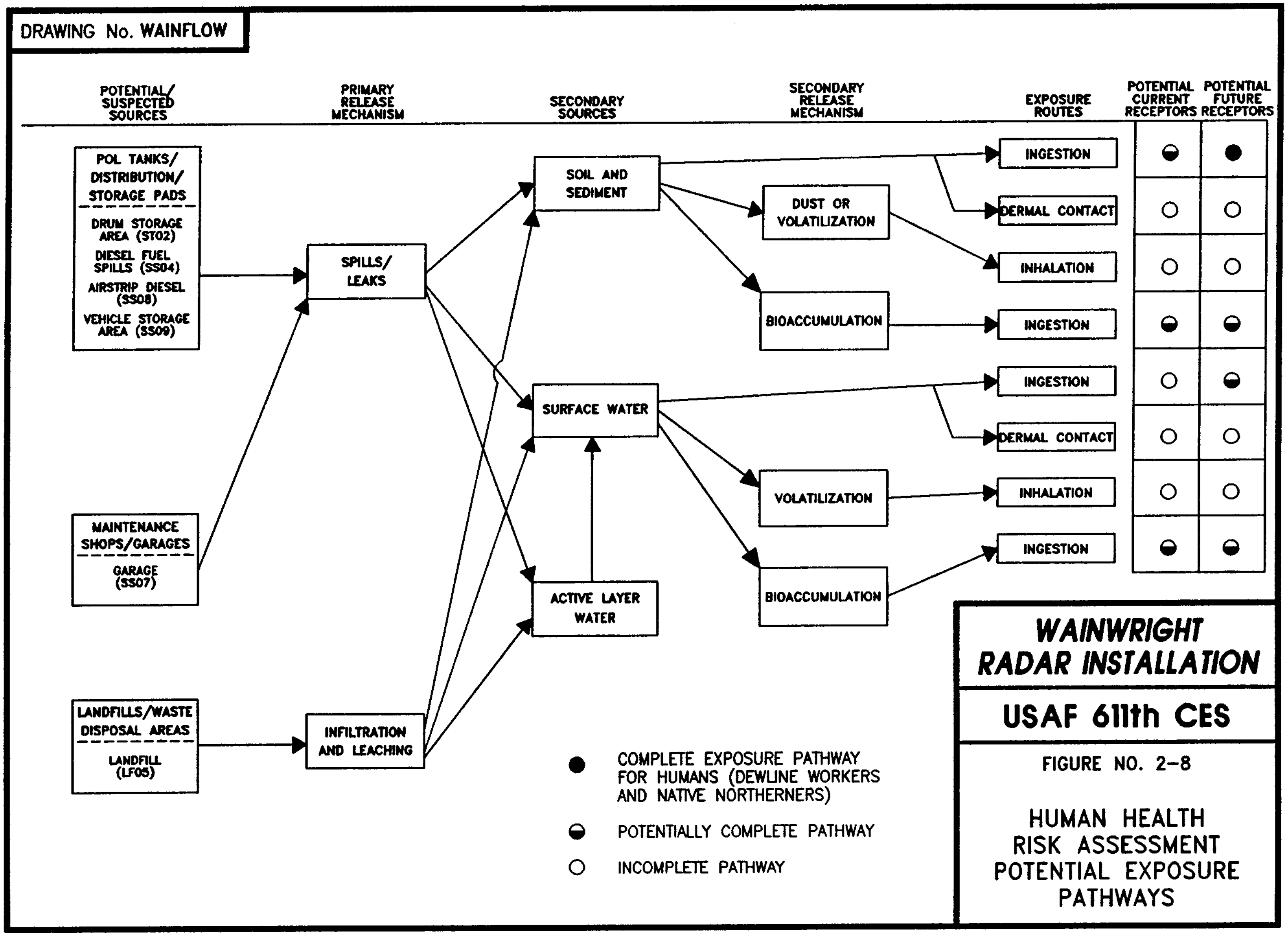
If one component of an exposure pathway does not exist, then exposure will not occur and there is no health risk. For example, if a shallow aquifer was contaminated with tetrachloroethene, but that aquifer was not used as a water supply, no exposure point would exist and the ground water ingestion pathway would not be complete.

The potential exposure pathways evaluated for the Wainwright human health risk assessment are presented in Figure 2-8 and Table 2-4, and are discussed in Sections 2.2.1.1 through 2.2.1.4.

2.2.1.1 Soil and Sediment Ingestion. Wainwright installation workers and residents of the community of Wainwright visiting the station could potentially be exposed to contaminated soil and sediment. The most likely exposure routes are incidental ingestion of soil and dermal absorption of contaminants in the soil. Site-specific characteristics will limit the magnitude, frequency, and duration of exposures to soil and sediment. The ground is covered with snow and ice, which eliminates soil or sediment exposure, for approximately nine months of the year. In the summer months when snow cover is generally absent, cool temperatures (30°F to 46°F) (University of Alaska 1978), keep both workers and villagers in heavy, long-sleeved clothing and gloves that eliminate dermal contact with, and hand-to-mouth transfer of, soil. Therefore, although both the incidental soil ingestion and dermal contact pathways are considered to be potentially complete, only incidental ingestion of soil or sediment will be evaluated further in this risk assessment.

The exposure assumptions used to evaluate the soil and sediment ingestion pathway are upper-bound residential scenario assumptions and, therefore, probably overestimate the true hazard or risk associated with this pathway. The purpose of using residential assumptions is to evaluate

THIS PAGE INTENTIONALLY LEFT BLANK


DRAWING No. WAINFLOW
POTENTIAL/ SUSPECTED SOURCES
PRIMARY RELEASE MECHANISM
SECONDARY SOURCES
SECONDARY RELEASE MECHANISM
EXPOSURE ROUTES
POTENTIAL CURRENT RECEPTORS
POTENTIAL FUTURE RECEPTORS
POL TANKS/ DISTRIBUTION/ STORAGE PADS
DRUM STORAGE AREA (ST02)
DIESEL FUEL SPILLS (SS04)
AIRSTRIP DIESEL (SS08)
VEHICLE STORAGE AREA (SS09)
MAINTENANCE SHOPS/GARAGES
GARAGE (SS07)
LANDFILLS/WASTE DISPOSAL AREAS
LANDFILL (LF05)
SPILLS/ LEAKS
INFILTRATION AND LEACHING
SOIL AND SEDIMENT
SURFACE WATER
ACTIVE LAYER WATER
DUST OR VOLATILIZATION
BIOACCUMULATION
VOLATILIZATION
BIOACCUMULATION
INGESTION
DERMAL CONTACT
INHALATION
INGESTION
INGESTION
DERMAL CONTACT
INHALATION
INGESTION
COMPLETE EXPOSURE PATHWAY FOR HUMANS (DEWLINE WORKERS AND NATIVE NORTHERNERS)
POTENTIALLY COMPLETE PATHWAY
INCOMPLETE PATHWAY
WAINWRIGHT RADAR INSTALLATION
USAF 611th CES
FIGURE NO. 2-8
HUMAN HEALTH RISK ASSESSMENT POTENTIAL EXPOSURE PATHWAYS

THIS PAGE INTENTIONALLY LEFT BLANK

TABLE 2-4. EXPOSURE PATHWAY ANALYSIS FOR WAINWRIGHT HUMAN HEALTH RISK ASSESSMENT

POTENTIALLY CONTAMINATED MEDIUM	POTENTIAL ROUTES OF EXPOSURE	POTENTIAL RECEPTORS	PATHWAY COMPLETE?	EXPOSED POPULATION ESTIMATE
Soil	Ingestion, dermal absorption	DEW Line workers, Wainwright residents	Ingestion, Yes Dermal Contact, No	584[a]
Sediments	Ingestion, dermal absorption	DEW Line workers, Wainwright residents	Ingestion, Yes Dermal Contact, No	584
Air	Inhalation of volatiles from soil or surface water or inhalation of fugitive dust	DEW Line workers, Wainwright residents	No, volatile concentrations in soil and surface water are very low; dust generation is not likely due to marshy vegetated landscape and high humidity, and snow and ice cover most of the year.	0
Surface Water	Incidental ingestion, dermal absorption	DEW Line workers, Wainwright residents	Maybe, drinking water supplies are either upgradient from installation or in unaffected areas; fishing occurs in unaffected areas; swimming does not occur onsite, however, incidental exposure may occur during installation operations or trespassing by native villagers.	584
Ground Water	Ingestion, dermal absorption	DEW Line workers, Wainwright residents	No, permafrost limits presence of ground water to shallow active layer that is not used for any purpose.	0

[a] Harcharek (1994).

the hazard or risk associated with future residential use of the Wainwright installation. Although the Air Force plans to convert the Wainwright radar installation to unmanned operation, it is possible at some time in the future that the installation may be closed and released for civilian use, in which case residential use of the installation may occur.

2.2.1.2 Inhalation. Wainwright installation workers and native northerners may be exposed to site contamination by inhalation of organic compounds volatilized from the soil or surface water, or windborne dust to which contamination has adsorbed. These exposure pathways are not considered complete for the Wainwright risk assessment because snow and ice cover the site for approximately nine months of the year and, during the summer months, the high humidity, vegetative cover, and thawing of surface and active layer water significantly limit the entrainment of dust particles. The generally low temperatures and high moisture content of the soil also tends to inhibit volatilization. The inhalation pathway will not be considered further in this risk assessment.

2.2.1.3 Water Ingestion. Surface water features, particularly those potentially contaminated by operations at the installation, are not likely to be used for drinking or other domestic purposes except on an incidental basis. This is because these surface water features are frozen for most of the year, and, therefore are not reliable sources of water for domestic or industrial use. Ingestion of surface water will, however, be considered a potentially complete exposure pathway to reflect the upper-bound potential future risk. Under current conditions, surface water at the installation is not used for domestic or other purposes. Fresh water for the area is obtained from a freshwater lake located approximately three miles north of the community of Wainwright. This lake is unaffected by contamination from the installation.

2.2.1.4 Ground Water. Permafrost limits the presence of ground water to the active layer, which thaws during the summer months. The water present in the active layer is not known to be used for any purpose; therefore, the ground water pathway will be eliminated from consideration in this risk assessment.

2.2.2 Migration and Fate of Chemicals of Concern

The COCs selected for Wainwright generally fall into three classes:

- refined and residual petroleum hydrocarbons (DRPH, GRPH, and RRPH);
- volatile and semi-volatile organic compounds (VOCs and SVOCs: 1,2-dichloroethane, tetrachlorethene, and bis(2-ethylhexyl)phthalate); and
- metals (barium, cadmium, manganese, vanadium, and zinc).

This section presents a summary of the migration and fate of each of these classes given the environmental conditions at Wainwright.

Once released to the environment, the COCs are immediately subject to several processes, including evaporation and volatilization, bulk flow, soil adsorption, dissolution in surface or active layer water, biodegradation, and photooxidation. The extent to which the COCs undergo each of these processes depends on their chemical and physical properties (e.g., K_{OC}, K_{OW}, water solubility, vapor pressure, and Henry's law constant), the volume released, soil flora, meteorological conditions, and soil and water organic carbon content.

The migration of petroleum hydrocarbons released to the gravel pads and tundra is expected to follow the rank order: GRPH > DRPH > RRPH. GRPH is generally considered to include hydrocarbons with carbon chain ranges from C5 to C12 that tend to be relatively mobile and less persistent than longer chain hydrocarbons. Depending on the length of time since a spill or leak occurred, the petroleum hydrocarbons observed in soil samples would be expected to be enriched in components that have carbon chain ranges greater than C10 or C11, have high K_{OC} and K_{OW} values, low vapor pressure and water solubility, and are not rapidly biodegradable. Petroleum components that fit this profile are higher molecular weight n-alkanes, mono- and poly-aromatics, and cycloalkanes. These components would tend to appear in laboratory analyses as diesel range or heavy oil range organics (DRPH and RRPH).

The migration of VOCs is expected to be rapid compared to the petroleum hydrocarbons. These compounds tend to have high vapor pressures which favor volatilization, high water solubility, and low K_{OC} and K_{OW} values. Therefore, the VOCs would tend to be highly mobile in the environment and dissipate rapidly after a spill or leak. In the results of field sampling, VOC concentrations would be expected to be fairly low depending on the time since the spill or leak occurred. The frigid conditions on the North Slope, however, would tend to reduce the mobility due to volatilization or evaporation.

The metals observed at Wainwright are probably of natural origin and not due to the operation of, or activities at, the radar installation. The presence of manganese in surface water samples is most often associated with landfill leachate since the anaerobic and acidic conditions inside of a landfill tends to release naturally occurring manganese from the soil. Metals will tend to be persistent and of low mobility in the environment.

In conclusion, the COCs observed at the Wainwright installation are generally expected to be fairly persistent and of low mobility. Exposure by contact with soils, primarily through accidental ingestion, is expected to predominate compared to exposure by inhalation.

2.2.3 Estimation of Chemical Intake

The exposure assessment for the Wainwright DEW Line installation required the development of site-specific assumptions because of the unique location and unmanned operation of the installation. This section of the report focuses on the exposure variables for which site-specific assumptions were made. These variables include:

- exposure frequency;
- exposure duration;
- ingestion of locally produced meat (e.g., caribou, fish, and birds);

- ingestion of locally produced vegetation (e.g., berries);
- soil ingestion rate; and
- rate of dermal contact with soil.

The exposure assumptions used in the human health risk assessment are presented in Table 2-5.

Three potential receptor groups will be evaluated for the Wainwright risk assessment: an adult assigned to maintenance work at the Wainwright installation (worker), an adult native of the North Slope of Alaska (native), and a native child (child). The native adult and child are considered to represent the RME that might occur at the installation. Because the Wainwright installation is close to the community of Wainwright and may be released for civilian use in the future, a child will be considered as a potentially exposed individual.

The estimation of chemical intake requires the evaluation of several exposure variables: exposure point concentration; exposure frequency; exposure duration; ingestion of locally produced meat, fish, and vegetation; soil ingestion; drinking water ingestion; dermal contact with soil; inhalation; and body weight. These exposure variables are discussed in the following sections.

TABLE 2-5. EXPOSURE ASSUMPTIONS FOR ESTIMATING CHEMICAL INTAKE

PARAMETER	DEW LINE WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN CHILD
Exposure Frequency - Soil Ingestion (days/year)	14	30	30
Exposure Frequency - Water Ingestion (days/year)	14	180	N/A
Exposure Duration (years)	10	55[a]	6[a]
Soil Ingestion Rate (mg/day)	50	100	200
Drinking Water Ingestion Rate (L/day)	2	2	N/A
Average Body Weight (kg)	70	70	15
Averaging Time (days)	25,550 (cancer)[b] 3,650 (noncancer)[c,d]	25,550 (cancer)[b] 20,075 (noncancer)[c]	2,190 (noncancer)[d]

N/A Not applicable; drinking water pathway evaluated for adult only.
[a] Exposure duration for water ingestion pathway is 55 years. For soil ingestion, exposure duration is 6 years as a child and 49 years as an adult.
[b] Averaging time for the evaluation of cancer risk by the soil and water ingestion pathways.
[c] Averaging time for the evaluation of noncancer hazard by the water ingestion pathway.
[d] Averaging time for the evaluation of noncancer hazard by the soil ingestion pathway.

2.2.3.1 Exposure Point Concentration. Based on the amount of analytical data available for the risk assessment of the Wainwright installation, and the requirement that the risk characterization be conducted individually for each of the six sites, only maximum concentrations of the COCs were used for exposure point concentrations. This approach yields a conservative upper-bound estimate of the ADD to which potential receptors may be exposed.

2.2.3.2 Exposure Frequency. The exposure frequency variable is an estimate of the amount of time a potential receptor may come in to contact with contaminated media. For the DEW Line worker, the exposure frequency estimate is based on the assumption that the Wainwright installation will require two maintenance visits per year, with each visit lasting fourteen days. Based on the assumption that the DEW Line maintenance work will require 12 hours per day, an estimate of the exposure frequency at the unmanned station would be:

$$12 \text{ hours/day} \times 1 \text{ day/24 hours} \times 28 \text{ days/year} = 14 \text{ days/year}$$

The exposure frequency estimate for a native adult or child of the North Slope is based on an estimate of the frequency with which the individual will visit a DEW Line installation. Such visits are most likely to occur at installations sited near a village or city. In this case, a conservative estimate of exposure would be expected to be similar to that of a DEW Line worker at an active facility, 4 hrs/day x 30 days per month x 1 day/24 hrs x 6 months of exposed soil = 30 days per year. In addition, this exposure frequency is expected to be similar for a future potential residential scenario.

The exposure frequency for water ingestion by the hypothetical native northern adult was conservatively estimated to be the 180 days/year that surface water would be available (i.e., not frozen).

2.2.3.3 Exposure Duration. The exposure duration variable is an estimate of the amount of time a potential receptor will remain at or near a DEW Line installation over a lifetime. For the DEW Line worker the exposure duration is an estimate of the maximum tour of duty at an installation. A conservative estimate of the duration of maintenance visits by an individual DEW Line worker to a particular installation is 10 years. For the potential native receptor, a conservative estimate of exposure duration is 55 years. EPA's default RME duration is 30 years; however, this is based on the overall U.S. population. Because the Alaskan natives are more likely to remain in their village for a longer period, 55 years was determined to be a more appropriate estimate based on best professional judgement.

2.2.3.4 Averaging Time. The averaging time represents the period of time over which exposure is averaged and is based on the assumption that intermittent exposure at a given contaminant concentration is equivalent to a continuous exposure at a lower concentration. For the DEW Line worker, the averaging time is based on the EPA default lifetime of 70 years for evaluation of carcinogens, and 10 years (equivalent to the exposure duration) for the evaluation of noncarcinogens. For the native northern adult, an averaging time of 70 years for carcinogens was also chosen. To evaluate exposure to noncarcinogens in soil and sediment for the native northern adult and child, an averaging time of 49 years as an adult and 6 years as a child was

used (to account for a 55 year total exposure). To evaluate the exposure of native northern receptors to noncarcinogens in water an averaging time of 55 years was used.

2.2.3.5 Ingestion of Locally Produced Meat, Fish, and Vegetation. The food supplies of DEW Line installation workers are largely imported from outside the area. Occasionally, a worker would be expected to ingest a locally caught fish or game animal, but the frequency and magnitude of this ingestion is expected to have a negligible effect on exposure to the COCs. Food supplies for the residents of Wainwright are also largely imported from outside the area, and the reliance on hunting and fishing for subsistence is decreasing substantially as the economy moves from subsistence to wage labor (Chance 1990). Inupiats, in general, have less time to hunt and fish than in the past. In the community of Wainwright, however, 50 percent of the households use subsistence activities to supply 10 or more meals per week (Harcharek 1994). Most of the hunting and fishing that is done occurs outside the village and away from the Wainwright DEW Line installation in areas unaffected by the installation. It is not likely that contamination observed at the installation has affected the mammals, birds, and vegetation that are collected for consumption. Therefore, the consumption of locally produced food is not likely to pose a significant risk of adverse health effects and will not be considered a complete exposure pathway. The ecological risk assessment, Section 3.0, presents a detailed assessment of risks to ecological receptors.

2.2.3.6 Soil Ingestion Rate. A conservative approach to estimating soil ingestion rate is to assume that the EPA default soil ingestion rates of 50 mg/day for workers (EPA 1991a) and 100 mg/day for adults in a residential setting (EPA 1989a). The EPA default soil ingestion rate for children is 200 mg/day (EPA 1989a, 1991a); this is the recommended value for the risk assessment.

2.2.3.7 Drinking Water Ingestion Rate. There are no circumstances at the Wainwright installation that would invalidate the EPA default adult drinking water ingestion rate of 2 L/day. Therefore, this is the recommended value for both workers and natives. However, in most, if not all, cases drinking water is imported from offsite so this may not be a route of potential exposure.

By convention (EPA 1989a), noncancer hazard and cancer risk associated with the drinking water pathway are evaluated for an adult receptor, not a child (Table 2-5). The basis for this approach is that the ratio of drinking water ingestion rate to body weight is assumed to remain relatively constant from childhood to adulthood.

2.2.3.8 Dermal Contact with Soil Rate. Because of the harsh North Slope weather, potential receptors (both workers and natives) are expected to be heavily clothed and gloved. Observations made by RI field personnel indicate that potential human receptors were heavily clothed during the months of the field investigations (August and September 1993). Therefore, dermal exposure to contaminated soils is considered negligible. In addition, the duties of installation workers that involve soil work (excavating, grading, etc.) are infrequent and are conducted in equipment with enclosed cabs. Thus a dermal contact rate does not appear to be necessary for the exposure assessment.

2.2.3.9 Inhalation Rate. The inhalation pathway is not complete (Section 2.2.1.2), so no estimate for this variable is necessary.

2.2.3.10 Body Weight. There are no circumstances at the Wainwright installation that would invalidate the EPA default adult body weight of 70 kg. Therefore, this is the recommended value for both workers and natives. The recommended body weight for children is the EPA default value of 15 kg.

2.2.4 Quantifying Exposure

For each complete, or potentially complete, exposure pathway at the Wainwright installation (soils ingestion, drinking water ingestion), the ADD for estimating noncancer hazard and the lifetime average daily dose (LADD) for estimating excess lifetime cancer risk were calculated. The equations used for the calculation of ADD and LADD are presented in Table 2-6. The exposure assumptions assigned to each variable in these equations are presented in Table 2-5. The estimates of ADD and LADD for the COCs at each site are presented in the risk characterization spreadsheets in Appendix A.

2.3 TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to weigh available evidence regarding the potential for particular contaminants to cause adverse effects in exposed individuals and to provide, where possible, an estimate of the relationship between the extent of exposure to a contaminant and the increased likelihood or severity of adverse effects or both. This is done separately for noncarcinogenic effects (Section 2.3.1) and carcinogenic effects (Section 2.3.2). Toxicity summaries are presented in Section 2.3.3.

Toxicity assessment for environmental contaminants is generally accomplished in two steps: hazard identification and dose-response assessment. Hazard identification is the process of determining whether exposure to an agent can cause an increase in the incidence of a particular adverse health effect (e.g., cancer, birth defects) and whether the adverse health effect is likely to occur in humans. Hazard identification involves characterizing the nature and strength of the evidence of causation. Dose-response evaluation is the process of quantitatively evaluating the toxicity information and characterizing the relationship between the dose of the contaminant administered or received and the incidence of adverse health effects in the exposed population. From this quantitative dose-response relationship, toxicity values [e.g., reference doses and slope factors (SFs)] are derived that can be used to estimate the incidence or potential for adverse effects as a function of human exposure to the agent. These toxicity values are used in the risk characterization step to estimate the likelihood of adverse effects occurring in humans at particular exposure levels.

TABLE 2-6. EQUATIONS USED FOR ESTIMATING POTENTIAL DOSE

EXPOSURE ROUTE	EQUATION	PARAMETER DEFINITIONS
Ingestion of Soil	Native Northern Adults/Children $ADD \text{ or } LADD\ (mg/kg/day) = \frac{C_s * CF * EF}{AT} \sum_{i=1}^{n} \frac{IR_i * ED_i}{BW_i}$ DEW Line workers: $ADD \text{ or } LADD\ (mg/kg/day) = \frac{C_s * CF * IR * EF * ED}{BW * AT}$	C_s = concentration in soil (mg/kg) CF = conversion factor (10^{-6} kg/mg) IR = ingestion rate (mg/day) EF = exposure frequency (days/year) ED = exposure duration (years) BW = body weight (kg) AT = averaging time (days/year x years)
Ingestion of Surface Water	$ADD \text{ or } LADD\ (mg/kg/day) = \frac{C_w * CF * IR * EF * ED}{BW * AT}$	C_w = concentration in surface water (μg/L) CF = conversion faction (10^{-3} mg/μg) IR = ingestion rate (L/day) EF = exposure frequency (days/year) ED = exposure duration (years) BW = body weight (kg) AT = averaging time (days/year x years)

2.3.1 Toxicity Assessment for Noncarcinogenic Effects

An RfD is the toxicity value used most often in evaluating noncarcinogenic effects resulting from exposures at contaminated sites. Various types of RfDs are available depending on the exposure route (oral or inhalation), the critical effect (developmental or other), and the length of exposure being evaluated (chronic, subchronic, or single event). The oral RfDs used to estimate the noncancer hazard associated with exposure to soils, sediments, and surface water at the Wainwright facility are presented in Table 2-7.

A chronic RfD is defined as an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. Chronic RfDs are developed specifically to be protective for long-term exposure to a compound. Chronic RfDs generally should be used to evaluate the potential noncancerous effects associated with exposure periods between 7 years (approximately 10 percent of a human lifetime) and a lifetime. Many chronic RfDs have been reviewed and verified by an intra-Agency RfD Workgroup and entered into EPA's IRIS database.

2.3.1.1 Concept of Threshold. For many noncancerous effects, protective mechanisms are believed to exist that must be overcome before the adverse effect is manifested. For example, where a large number of cells perform the same or similar function, the cell population may have to be significantly depleted before the adverse effect is seen. As a result, a range of exposures exists from zero to some finite level that can be tolerated by the organism with essentially no chance of expression of adverse effects. In developing a toxicity value for evaluating noncancerous effects (i.e., an RfD), the approach is to identify the upper bound of this tolerance range (i.e., the maximum subthreshold level). Because variability exists among humans, attempts are made to identify a subthreshold level that protects sensitive individuals in the population. For most chemicals, this level can only be estimated; the RfD incorporates uncertainty factors (UFs) indicating the degree of extrapolation used to derive the estimated value. RfD summaries in IRIS also contain a statement expressing the overall confidence that the evaluators have in the RfD (high, medium, or low). The RfD is generally considered to have uncertainty spanning an order of magnitude or more, and therefore the RfD should not be viewed as a strict scientific demarcation between levels that are toxic and nontoxic.

2.3.2 Toxicity Assessment For Carcinogenic Effects

An SF and the accompanying weight-of-evidence determination are the toxicity data most commonly used to evaluate potential human carcinogenic risks. The methods EPA uses to derive these values are outlined below. Additional information can be obtained by consulting EPA's *Guidelines for Carcinogen Risk Assessment* (EPA 1986a) and IRIS Background Document #2 (IRIS 1994). The SFs for the COCs at Wainwright are presented in Table 2-8.

2.3.2.1 Concept of Nonthreshold Effects. Risk evaluation based on the presumption of a dose-response threshold is generally thought to be inappropriate for carcinogenesis. In the evaluation of carcinogens, EPA assumes that a small number of molecular events can evoke changes in a single cell and lead to uncontrolled cellular proliferation and eventually to clinical

TABLE 2-7. TOXICITY CRITERIA FOR NONCANCER EFFECTS OF THE CHEMICALS OF CONCERN FOR WAINWRIGHT

CHEMICAL	ORAL REFERENCE DOSE (RfD) (mg/kg-day)	TARGET ORGAN OR CRITICAL EFFECT (species)[a]	UNCERTAINTY FACTOR[b]	SOURCE ORAL RfD[c]
Barium	0.07	NOAEL for increased blood pressure (humans)	3	IRIS
Bis(2-ethylhexyl)phthalate	0.02	liver effects (guinea pig)	1,000	IRIS
Cadmium	0.001	kidney (humans)	10	IRIS
Dichloroethane, 1,2-	NA	NA	NA	NA
DRPH	0.08[d]	liver effects (mice)	10,000	ECAO
GRPH	0.2[d]	decreased body weight (rats)	1,000	ECAO
Manganese (water)	0.005	CNS effects (humans)	1	IRIS
RRPH	0.08[d]	liver effects (mice)	10,000	ECAO
Tetrachloroethene	0.01	liver effects (mice)	1,000	IRIS
Vanadium	0.007	NA	100	HEAST
Zinc	0.3	decreased erythrocyte SOD (humans)	3	IRIS

a A target organ is the organ apparently most sensitive to the toxicity of a chemical. A critical effect is reported when EPA has not identified a target organ for the toxicity of a given chemical.

b The uncertainty factors used to develop oral reference doses are generally applied in multiples of 10 to account for shortcomings in the toxicological database. The greater the uncertainty factor, the lower the confidence level in the RfD. Factors of 10 are applied to account for human variability in toxic response, extrapolation from animal studies to humans, extrapolation of short-term exposures to long-term exposures, and for the extrapolation of a lowest-observed adverse effect level (LOAEL) to a no observed adverse effect level (NOAEL).

c Sources of oral RfD values are IRIS (Integrated Risk Information System), HEAST (Health Effects Assessment Summary Tables), or ECAO (The Environmental Criterion Assessment Office of EPA).

d Oral RfD values for DRPH, GRPH, and RRPH are based on (EPA 1992b) and are considered provisional RfDs.

NA Not available.

TABLE 2-8. TOXICITY VALUES FOR THE CARCINOGENICITY OF THE CHEMICALS OF CONCERN AT WAINWRIGHT

CHEMICAL	WEIGHT-OF-EVIDENCE (WOE)	TUMOR TYPE (species)	ORAL SLOPE FACTOR (kg-day/mg)	ORAL SLOPE FACTOR SOURCE[a]
Barium	NA	NA	NA	IRIS
Bis(2-ethylhexyl)phthalate	B2	liver carcinoma/adenoma (mouse)	0.014	IRIS
Cadmium	NA[b]	NA	NA	IRIS
Dichloroethane, 1,2-	B2	hemangiosarcomas (rat)	0.091	IRIS
DRPH	NA	NA	NA	NA
GRPH	C	liver adenoma/carcinoma (mouse)	0.0017	ECAO
Manganese (water)	NA	NA	NA	IRIS
RRPH	NA	NA	NA	NA
Tetrachloroethene	C-B2	not specified	0.052	ECAO
Vanadium	NA	NA	NA	IRIS
Zinc	NA	NA	NA	IRIS

a IRIS, Integrated Risk Information System; HEAST, Health Effects Assessment Summary Tables; ECAO, Environmental Criterion Assessment Office of EPA.
b There is inadequate evidence of carcinogenicity for this analyte by the oral route.
NA Not available.

state of disease (cancer). This hypothesized mechanism for carcinogenesis is referred to as "nonthreshold" because all levels of exposure pose some probability of generating a carcinogenic response. Thus, no dose is thought to be risk-free, and an effect threshold cannot be estimated.

For carcinogenic effects, EPA uses a two-part evaluation in which the substance first is assigned a weight-of-evidence classification, then an SF is calculated.

2.3.2.2 Assigning a Weight-of-Evidence. In the first step of the evaluation, the carcinogenicity data are evaluated to determine the likelihood that the agent is a human carcinogen. The evidence is characterized separately for human studies and animal studies as sufficient, limited, inadequate, no data, or evidence of no effect. The characterizations of these two types of data are combined and, based on the extent to which the agent has been shown to be a carcinogen in experimental animals, humans, or both, the agent is given a provisional weight-of-evidence classification. EPA scientists then adjust the provisional classification upward or downward, based on other supporting evidence of carcinogenicity.

The EPA classification system for weight-of-evidence is shown in Table 2-9.

2.3.2.3 Generating a Slope Factor. For chemicals classified as known or probable human carcinogens, a toxicity value that defines quantitatively the relationship between dose and response (i.e., the SF) is calculated. SFs are typically calculated for potential carcinogens in classes A, B1, and B2. Quantitative estimation of SFs for the chemicals in class C is done on a case-by-case basis.

TABLE 2-9. EPA WEIGHT-OF-EVIDENCE CLASSIFICATION SYSTEM FOR CARCINOGENICITY

GROUP	DESCRIPTION
A	Human carcinogen.
B1 or B2	Probable human carcinogen.
	B1 indicates that limited human data are available.
	B2 indicates sufficient evidence in animals and inadequate or no evidence in humans.
C	Possible human carcinogen.
D	Not classifiable as to human carcinogenicity.
E	Evidence of noncarcinogenicity for humans.

Generally, the SF is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical over a lifetime. The SF is used in risk assessments to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen. SFs should always be accompanied by the weight-of-evidence classification to indicate the strength of the evidence that the agent is a human carcinogen.

2.3.2.4 Identifying the Appropriate Data Set. In deriving SFs, the available information about a chemical is evaluated, and an appropriate data set is selected. In choosing appropriate data sets, human data of high quality are preferable to animal data. If animal data are used, the species that responds most similarly to humans (with respect to factors such as metabolism, physiology, and pharmacokinetics) is preferred. When no clear choice is possible, the most sensitive species is given the greatest emphasis. Occasionally, in situations where no single study is judged most appropriate yet several studies collectively support the estimate, the geometric mean of estimates from all studies may be adopted as the SF. This practice ensures the inclusion of all relevant data.

2.3.2.5 Extrapolating to Lower Doses. Because risk at low exposure levels is difficult to measure directly either by animal experiments or by epidemiologic studies, the development of an SF generally entails applying a model to the available data set and using the model to extrapolate from the relatively high doses administered to experimental animals (or the exposures noted in epidemiologic studies) to the lower exposure levels expected for human contact in the environment.

A number of mathematical models and procedures have been developed to extrapolate from carcinogenic responses observed at high doses to responses expected at low doses. Different extrapolation methods may provide a reasonable fit to the observed data but may lead to large differences in the projected risk at low dose.

In general, after the data are fit to the appropriate model, the upper 95th percent confidence limit of the slope of the resulting dose-response curve is calculated. This value is known as the SF and represents an upper 95th percent confidence limit on the probability of a response per unit intake of a chemical over a lifetime (i.e., there is only a five percent chance that the probability of a response could be greater than the estimated value on the basis of the experimental data and model used). In some cases, SFs based on human dose-response data are based on the "best" estimate instead of the upper 95th percent confidence limits. Because the dose-response curve generally is linear only in the low-dose region, the SF estimate only holds true for low doses. Information concerning the limitations on use of SFs can be found in IRIS.

2.3.2.6 Summary of Dose-Response Parameters. Toxicity values for carcinogenic effects can be expressed in several ways. The SF is generally considered to be the upper 95th percent confidence limit of the slope of the dose-response curve and is expressed as (mg/kg-day)$^{-1}$. Thus:

$$\text{Slope factor} = \text{risk per unit dose} = \text{risk per mg/kg-day}$$

Where data permit, SFs listed in IRIS are based on absorbed doses, although many of them have been based on administered doses.

2.3.3 Summaries of the Toxicity of the Contaminants of Concern

Tables 2-7 and 2-8 present noncancer and cancer toxicity criteria (RfDs and oral SFs, respectively) for the COCs. The toxicological properties of the COCs and the toxicological basis of the health effects criteria listed in Tables 2-7 and 2-8 are discussed in Appendix B.

2.4 RISK CHARACTERIZATION

In the risk characterization, the toxicity and exposure assessments are summarized and integrated into quantitative and qualitative expressions of risk. To characterize potential noncancerous effects, comparisons are made between projected intakes of substances and toxicity values (e.g., the reference dose); to characterize potential carcinogenic effects, probabilities that an individual will develop cancer over a lifetime of exposure are estimated from projected intakes and chemical-specific dose-response information (e.g., the SF). Major assumptions, scientific judgements and, to the extent possible, estimates of the uncertainties embodied in the assessment are also presented. In this section, methods of quantifying risks are discussed and applied to individual sites on the Wainwright installation.

2.4.1 Quantifying Risks

This section describes steps for quantifying risk or hazard indices for both carcinogenic and noncancerous effects to be applied to each exposure pathway analyzed. The first two subsections cover procedures for individual substances and are followed by a subsection on procedures for quantifying risks associated with simultaneous exposures to several substances.

2.4.1.1 Risks from Individual Substances - Carcinogenic Effects. For carcinogens, risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential carcinogen (i.e., incremental or excess individual lifetime cancer risk). The guidelines provided in this section are consistent with EPA (1986b). For some carcinogens, there may be sufficient information on mechanism of action that a modification of the approach outlined below is warranted. Alternative approaches may be considered in consultation with ECAO on a case-by-case basis.

The SF converts estimated daily intakes averaged over a lifetime of exposure directly to incremental risk of an individual developing cancer. Because relatively low intakes (compared to those experienced by test animals) are most likely from environmental exposures, it generally can be assumed that the dose-response relationship will be linear in the low-dose portion of the multistage model dose-response curve. Under this assumption the SF is a constant, and risk will be directly related to intake. Thus, the linear form of the carcinogenic risk equation is usually applicable for estimating cancer risks. This linear low-dose equation is described below.

LINEAR LOW-DOSE CANCER RISK EQUATION

Risk = LADD x SF

where:

Risk = a unitless probability (e.g., 2×10^{-5}) of an individual developing cancer;

LADD = lifetime average daily dose averaged over 70 years (mg/kg-day); and

SF = slope factor, expressed in $(\text{mg/kg-day})^{-1}$

Because the SF is often an upper 95th percentile confidence limit of the probability of response based on experimental animal data used in the multistage model, the carcinogenic risk estimate will generally be an upper-bound estimate. This means that the "true risk" will probably not exceed the risk estimate derived through use of this model and is likely to be less than predicted.

2.4.1.2 Noncancer Hazards from Individual Substances - Noncancerous Effects. The measure used to describe the potential for noncancerous toxicity to occur in an individual is not expressed as the probability of an individual suffering an adverse effect. EPA does not at the present time use a probabilistic approach to estimate the potential for noncancerous health effects. Instead, the potential for noncancerous effects is evaluated by comparing an exposure level over a specified time period (e.g., some fraction of a lifetime) with an RfD derived for a similar exposure period. This ratio of exposure to toxicity is called an HQ.

The noncancer HQ assumes there is a level of exposure (i.e., the RfD) below which it is unlikely for even sensitive populations to experience adverse health effects. If the exposure level (ADD) exceeds this threshold (i.e., if ADD/RfD exceeds unity), there may be concern for potential noncancer effects. As a rule, the greater the value of ADD/RfD above unity, the greater the level of concern. Ratios of ADD/RfD should not be interpreted as statistical probabilities; a ratio of 0.001 does not mean that there is a one in one thousand chance of the effect occurring. Further, it is important to emphasize that the level of concern does not increase linearly as the RfD is approached or exceeded because RfDs do not have equal accuracy or precision and are not based on the same severity of toxic effects. Thus, the slopes of the dose-response curve in excess of the RfD can range widely depending on the substance.

NONCANCER HAZARD QUOTIENT

Noncancer Hazard Quotient = ADD/RfD

where:

ADD = average daily dose (or intake);

RfD = reference dose

ADD and RfD are expressed in the same units and represent the same exposure period (e.g., chronic, subchronic, or short-term).

2.4.1.3 Aggregate Risks for Multiple Substances. Estimating risk or hazard potential by considering one chemical at a time might significantly underestimate the risks associated with simultaneous exposures to several substances. To assess the overall potential for cancer and

noncancer effects posed by multiple chemicals, EPA has developed *Guidelines for the Health Risk Assessment of Chemical Mixtures* (EPA 1986b) that can also be applied in the case of simultaneous exposures to several chemicals from a variety of sources by more than one exposure pathway. Information on specific mixtures, however, is rarely available. Even if such data exist, they are often difficult to use. Monitoring for "mixtures" or modeling the movement of mixtures across space and time presents significant technical problems given the likelihood that individual components will behave differently in the environment (i.e., fate and transport).

Although the calculation procedures differ for carcinogenic and noncarcinogenic effects, both sets of procedures assume dose additivity in the absence of information on specific mixtures.

Carcinogenic effects. The cancer risk equation described below is used to estimate the incremental individual lifetime cancer risk for simultaneous exposure to several carcinogens and is based on EPA's risk assessment guidelines. This equation represents an approximation of the precise equation for combining risks that accounts for the joint probabilities of the same individual developing cancer as a consequence of exposure to two or more carcinogens. The difference between the precise equation and the approximation described in the equation below is negligible for total cancer risks less than 0.1. Thus, the simple additive equation is appropriate for most risk assessments.

CANCER RISK EQUATION FOR MULTIPLE SUBSTANCES

$$Risk_T = \Sigma\, Risk_i$$

where:

$Risk_T$ = the total cancer risk, expressed as a unitless probability; and

$Risk_i$ = the risk estimate for the i^{th} substance.

The risk summation techniques described in the cancer risk equation above assume that intakes of individual substances are small. They also assume independence of action by the compounds involved (i.e., there are no synergistic or antagonistic chemical interactions and all chemicals produce the same effect, i.e., cancer). If these assumptions are incorrect, over- or under-estimation of the actual multiple-substance risk could result.

A separate total cancer risk for each exposure pathway is calculated by summing the substance-specific cancer risks. Resulting cancer risk estimates should be expressed using one significant figure only.

There are several limitations to this approach. First, because each SF is an upper 95th percentile estimate of potency, and because upper 95th percentiles of probability distributions are not strictly additive, the total cancer risk estimate might become artificially more conservative as risks from a number of different carcinogens are summed. If one or two carcinogens drive the risk, however, this problem is not of concern. Second, it often will be the case that substances with different weights of evidence for human carcinogenicity are included. The cancer risk equation for multiple substances sums all carcinogens equally, giving as much weight to class B or C as to class A carcinogens. In addition, SFs derived from animal data will be given the same weight

as SFs derived from human data. Finally, the action of two different carcinogens might not be independent.

Noncancerous effects. To assess the overall potential for noncancerous effects posed by more than one chemical, a hazard index approach has been developed based on EPA's *Guidelines for Health Risk Assessment of Chemical Mixtures* (EPA 1986b). This approach assumes that simultaneous subthreshold exposures to several chemicals could result in an adverse health effect. It also assumes that the magnitude of the adverse effect will be proportional to the sum of the ratios of the subthreshold exposures. The hazard index is equal to the sum of the HQs. When the hazard index exceeds unity, there may be concern for potential health effects. Any single chemical with an exposure level greater than the toxicity value will cause the hazard index to exceed unity and, for multiple chemical exposures, the hazard index can also exceed unity even if no single chemical exposure exceeds its RfD. The equation used to determine noncancer hazard index is as follows:

NONCANCER HAZARD INDEX

$$\text{Hazard Index} = ADD_1/RfD_1 + ADD_2/RfD_2 + \ldots + ADD_i/RfD_i$$

where:

ADD_i = average daily dose (or intake) for the i^{th} toxicant;

RfD_i = reference dose for the i^{th} toxicant; and

ADD and RfD are expressed in the same units and represent the same exposure period (i.e., chronic, subchronic, or shorter-term).

Where appropriate, a separate chronic hazard index can be calculated from the ratios of the chronic daily intake (CDI) to the chronic RfD for individual chemicals as described below.

CHRONIC NONCANCER HAZARD INDEX

$$\text{Chronic Hazard Index} = LADD_1/RfD_1 + LADD_2/RfD_2 + \ldots + LADD_i/RfD_i$$

where:

$LADD_i$ = lifetime average daily dose for the i^{th} toxicant in mg/kg-day, and

RfD_i = chronic reference dose for the i^{th} toxicant in mg/kg-day.

There are several limitations to this approach. As mentioned earlier, the level of concern does not increase linearly as the RfD is approached or exceeded because the RfDs do not have equal accuracy or precision and are not based on the same severity of effect. Moreover, HQs are combined for substances with RfDs based on critical effects of varying toxicological significance. Also, it will often be the case that RfDs of varying levels of confidence that include different uncertainty adjustments and modifying factors will be combined (e.g., extrapolation from animals to humans, from LOAELs to no observed adverse effect levels (NOAELs), from one exposure duration to another).

Another limitation with the hazard index approach is that the assumption of dose additivity is most properly applied to compounds that induce the same effect by the same mechanism of

action. Consequently, application of the hazard index equation to a number of compounds that are not expected to induce the same type of effects or that do not act by the same mechanism could overestimate the potential for effects, although such an approach is appropriate at a screening level. This possibility is generally not of concern if only one or two substances are responsible for driving the hazard index above unity. If the hazard index is greater than unity as a consequence of summing several HQs of similar value, it would be appropriate to segregate the compounds by effect and by mechanism of action and to derive separate hazard indices for each group.

2.4.2 Site-Specific Risk Characterization

Soil and Sediment Exposures. The quantification of noncancer hazard and excess lifetime cancer risk associated with the soil ingestion pathway at Wainwright was based on analytical data from soil and sediment samples collected within the interval from ground surface to permafrost. No attempt was made to segregate surface soil samples from subsurface samples in the risk characterization.

The noncancer hazard and excess lifetime cancer risk associated with the ingestion of soil or sediment containing COCs has been estimated separately for a native northern adult, native northern child, and DEW Line worker. The noncancer hazard and the excess lifetime cancer risk associated with the ingestion of soil or sediment containing COCs has been estimated for a hypothetical native northerner based on six years of exposure as a child and 49 years of exposure as an adult. For the DEW Line worker, cancer risk has been estimated based on ten years of exposure averaged over a default lifetime of 70 years. Noncancer hazard for the DEW Line worker was based on a 10 year exposure.

Surface Water Exposures. The noncancer hazard and the excess lifetime cancer risk associated with the ingestion of surface water containing COCs has been estimated based on a native northern adult and a DEW Line worker. A native northern child receptor was not considered because, unlike exposure to soil, which is expected to be greater in a child than in an adult, the ratio of drinking water ingestion rate to body weight is assumed to be relatively constant from childhood to adulthood. A greater number of years is spent as an adult, so estimating hazard or risk for water ingestion based on an adult is a more conservative approach. The exposure duration estimate for the DEW Line worker was 10 years and for the native northern adult was 55 years. Exposures were averaged over 10 years for DEW Line worker exposure to noncarcinogens, and 55 years for native northern adult exposure to noncarcinogens. Exposures were averaged over 70 years for both receptor groups to characterize the risk associated with exposure to carcinogens in surface water.

Ingestion of surface water at the Wainwright installation is not considered to be a complete pathway under a current use scenario; the installation is being automated for unmanned operation and is located three to four miles from the community of Wainwright. Under a future use scenario, however, it is possible that the buildings could be used for residences or additional residential structures could be erected at the installation. The future residents could be either DEW Line workers or native northerners. The residents of the community of Wainwright currently receive their domestic water from a fresh-water lake located two to three miles north of the city.

This water is transported by a pipeline to a treatment facility in the city and then distributed by truck. Therefore, because sources of water may change in the future, potential ingestion of surface water at the installation will be evaluated for the DEW Line worker and native northern adult under a future use exposure scenario only.

Table 2-10 contains a site-by-site summary of the COCs in each medium, and the noncancer hazard and excess lifetime cancer risk associated with exposure to the soils/sediments and surface water. Table 2-10 does not include sites where no COCs were identified [Drum Storage Area (ST02) and Airstrip Diesel (SS08)]. COCs without toxicity data are not included on Table 2-10, but are discussed in Section 2.1.5. Appendix A contains the spreadsheets used to calculate the noncancer hazard and excess lifetime cancer risk estimates presented in Table 2-10.

Risk Characterization of Petroleum Hydrocarbons. Petroleum hydrocarbons represent a primary source of contamination at the Wainwright installation. The laboratory analysis of soil, sediment, and surface water samples revealed the presence of DRPH, GRPH, and RRPH. In the process of characterizing the risk associated with exposure to these compounds, it was necessary to apply the provisional RfDs and the SF developed by EPA for petroleum hydrocarbons (EPA 1992b). These provisional RfDs provide the best available tool for characterizing the risk associated with exposure to the petroleum hydrocarbons. The RfD for JP-4 presented in EPA (1992b) was assumed to represent DRPH and RRPH, and the RfD and SF for unleaded gasoline were assumed to represent GRPH.

The noncancer hazard associated with exposure to DRPH, GRPH, and RRPH was estimated by dividing the compound- and site-specific ADD by the appropriate provisional RfD (EPA 1992b). The excess lifetime cancer risk associated with exposure to GRPH was estimated by multiplying the compound- and site-specific LADD by the SF for unleaded gasoline (EPA 1992b).

Although the provisional RfDs and SF represent the best available numerical estimate of toxicity, there is a significant amount of uncertainty associated with their use at the Wainwright installation. The RfDs and SF are based on studies in mice and rats that used inhalation as the route of exposure; whereas for this risk assessment, exposure of humans by the ingestion route is being evaluated. Furthermore, in the absence of a more thorough study to compare the DRPH, GRPH, and RRPH to known petroleum refinery streams, it is not clear how well the provisional values represent the toxicity of diesel and gasoline in humans.

Risk Characterization of Chemicals Without RBSLs and ARARs. Chemicals detected above background levels without RBSLs or ARARs are evaluated in Section 2.1.5 (page 2-16). Based on the information in that section, and the relatively low levels detected at the sites, these chemicals are not expected to pose a health risk.

2.4.2.1 Drum Storage Area (ST02).

Soil and Sediments. No COCs were identified for the soil at the Drum Storage Area (ST02) (Table 2-10). This does not indicate that exposure to chemicals in the soil at the site is without

TABLE 2-10. SUMMARY OF NONCANCER HAZARD AND EXCESS LIFETIME CANCER RISK FOR WAINWRIGHT

SITE	MEDIUM	NONCANCER COCs[a]	CARCINOGENIC COCs[a]	NONCANCER HAZARD[c]			EXCESS LIFETIME CANCER RISK[d]		
				DEW LINE WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN ADULT/CHILD	DEW LINE WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN ADULT/CHILD
Drum Storage Area (ST02)	Soil	NONE[b]	NONE[b]	—	—	—	—	—	—
	Surface Water	NONE	NONE	—	—	—	—	—	—
Diesel Fuel Spills (SS04)	Soil	DRPH GRPH	GRPH	0.002	ND[e]	0.08	8×10^{-8}	ND	4×10^{-8}
	Surface Water	NONE	NONE	—	—	—	—	—	—
Landfill (LF05)	Soil	GRPH Cadmium	GRPH	0.002	ND	0.02	1×10^{-9}	ND	6×10^{-8}
	Surface Water	NONE	1,2-Dichloroethane	ND	ND	ND	9×10^{-8}	**8×10^{-6}**	ND
Garage (SS07)	Soil	DRPH GRPH RRPH Tetrachloroethene	GRPH Tetrachloroethene	0.07	ND	3	3×10^{-9}	ND	1×10^{-7}
	Surface Water	Bis(2-ethylhexyl)phthalate	bis(2-ethylhexyl)phthalate 1,2-Dichloroethane	<0.001	0.01	ND[f]	6×10^{-8}	**5×10^{-6}**	ND
Airstrip Diesel (SS08)	Soil	NONE	NONE	—	—	—	—	—	—
	Surface Water	NONE	NONE	—	—	—	—	—	—
Vehicle Storage Area (SS09)	Soil	NONE	NONE	–	–	–	–	–	–
	Surface Water	Barium Manganese Vanadium Zinc	1,2-Dichloroethane	0.9	11	ND	2×10^{-8}	**2×10^{-6}**	ND

TABLE 2-10. SUMMARY OF NONCANCER HAZARD AND EXCESS LIFETIME CANCER RISK FOR WAINWRIGHT (CONTINUED)

ND Not determined (soil ingestion pathway evaluated for DEW Line worker and the native northern adult/child combination only; surface water ingestion evaluated for DEW Line worker and the native northern adult only. See sections 2.2.2.4 and 2.2.2.7 for further explanation).

BOLD Text indicates exceedance of regulatory benchmarks: HQ > 1, CR > 1 x 10^{-6}.

a All COCs are listed together regardless of whether they contribute to the hazard index, cancer risk, or both.

b None, no COCs selected.

c Hazard index, noncancer hazard index. The hazard index is the sum of the HQs for all of the COCs associated with a given medium, pathway, and receptor group.

d Cancer risk, excess lifetime cancer risk. The cancer risk is the sum of the excess lifetime cancer risks for all of the carcinogenic COCs associated with a given medium, pathway, and receptor group.

e Children are assumed to have a soil ingestion rate greater than that for adults. Therefore, under a residential scenario, the estimates of noncancer hazard and cancer risk associated with soil ingestion are estimated for a combined adult and child receptor only. This estimate is considered a conservative upper bound on the true hazard or risk.

f Drinking water ingestion, unlike soil ingestion, is evaluated for an adult receptor but not a child receptor because adults are assumed to have a longer exposure duration at a greater water ingestion rate. Therefore, the hazard or risk estimated will represent an upper bound, conservative estimate. For soil ingestion, the child soil ingestion rate is assumed to exceed that for adults. Therefore, a combination of the adult and child receptor groups is used to evaluate soil ingestion risk and hazard.

health risk; however, the concentrations measured were lower than the concentrations considered acceptable under Region 10 guidance (EPA 1991a) or federal ARARs.

Surface Water. No surface water bodies were identified at the site; therefore, no evaluation of noncancer hazard or excess lifetime cancer risk associated with ingestion of surface water was conducted.

2.4.2.2 Diesel Fuel Spills (SS04).

Soil and Sediment. The noncancer hazard associated with the ingestion of soil at Diesel Fuel Spills (SS04) by a hypothetical native northern adult/child is 0.8, and by a DEW Line worker is 0.002, based on the maximum concentrations of the COCs (Tables 2-10 and A-1). The presence of DRPH and GRPH accounts entirely for the quantifiable noncancer hazard for these receptor/pathway combinations.

The excess lifetime cancer risk associated with the ingestion of soil at the site by a hypothetical native northern adult/child is 4×10^{-8}, and by a DEW Line worker is 8×10^{-10}, based on the maximum concentrations of the COCs (Tables 2-10 and A-2). The presence of GRPH accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

Surface Water. No COCs were identified for the surface water at the site (Table 2-10). This does not indicate that exposure to the chemicals in the surface water at the site is without health risk; however, the concentrations measured were lower than the concentrations considered acceptable under Region 10 guidance (EPA 1991a) or federal ARARs.

2.4.2.3 Landfill (LF05).

Soil and Sediments. The noncancer hazard associated with the ingestion of soil at the Landfill (LF05) by a hypothetical native northern adult/child is 0.02, and by a DEW Line worker is 0.002, based on the maximum concentration of the COCs (Tables 2-10 and A-3). The presence of GRPH and cadmium accounts entirely for the quantifiable noncancer hazard for these receptor/pathway combinations.

The excess lifetime cancer risk associated with the ingestion of soil at the site by a hypothetical native northern adult/child is 6×10^{-8}, and by a DEW Line worker is 1×10^{-9}, based on the maximum concentration of the COC (Tables 2-10 and A-4). The presence of GRPH accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

Surface Water. No COCs were identified for the surface water at the Landfill (LF05) (TAble 2-10). This does not indicate that exposure to chemicals in the surface water at the site is without health risk; however, the concentrations measured were lower than the concentrations considered acceptable under Region 10 guidance (EPA 1991a) or federal ARARs.

The excess lifetime cancer risk associated with the ingestion of surface water at the site by a hypothetical native northern adult is 8×10^{-6}, and by a DEW Line worker is 9×10^{-8}, based on the maximum concentration of the COC (Tables 2-10 and A-6). The presence of 1,2-

dichloroethane accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

2.4.2.4 Garage (SS07).

Soil and Sediment. The noncancer hazard associated with the ingestion of soil or sediment at the Garage (SS07) by a hypothetical native northern adult/child is 3, and by a DEW Line worker is 0.07, based on the maximum concentrations of the COCs (Tables 2-10 and A-7). The presence of DRPH, GRPH, RRPH, and tetrachloroethene accounts for the quantifiable noncancer hazard for these receptor/pathway combinations.

The excess lifetime cancer risk associated with the ingestion of soil or sediment at the site by a hypothetical native northern adult/child is 1×10^{-7}, and by a DEW Line worker is 3×10^{-9}, based on the maximum concentrations of the COCs (Tables 2-10 and A-8). The presence of GRPH and tetrachloroethene accounts for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

Surface Water. The noncancer hazard associated with the potential future ingestion of surface water at the site by a hypothetical native northern adult is 0.01, and by a DEW Line worker is less than 0.001, based on the maximum concentrations of the COC (Tables 2-10 and A-9). The presence of bis(2-ethylhexyl)phthalate accounts entirely for the quantifiable noncancer hazard for these receptor/pathway combinations.

The excess lifetime cancer risk associated with the potential future ingestion of surface water at the site by a hypothetical native northern adult is 5×10^{-6}, and by a DEW Line worker is 6×10^{-8}, based on the maximum concentrations of the COCs (Tables 2-10 and A-10). The presence of bis(2-ethylhexyl)phthalate and 1,2-dichloroethane accounts entirely for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

2.4.2.5 Airstrip Diesel (SS08).

Soil and Sediment. No COCs were identified for the sediment at the Airstrip Diesel (SS08) because no sediment sample contained any chemical at a concentration exceeding the detection limit. This does not indicate that exposure to the sediment at the Airstrip Diesel is without health risk; however, any risk that may be associated with exposure to sediment at the site is probably not of significance for humans.

Surface Water. No COCs were identified for the surface water at the site because no surface water sample analyzed contained any chemical at a concentration exceeding the detection limit. This does not indicate that exposure to the surface water at the site is without health risk, however any risk that may be associated with exposure to surface water at the site is probably not of significance for humans.

2.4.2.6 Vehicle Storage Area (SS09).

Soil and Sediment. No COCs were identified for the soil at the Vehicle Storage Area (SS09) (Table 2-10). This does not indicate that exposure to chemicals in the soil at the site is without health risk; however, the concentrations measured were lower than the concentrations considered acceptable under Region 10 guidance (EPA 1991a) or federal ARARs.

Surface Water. The noncancer hazard associated with the potential future ingestion of surface water at the site by a hypothetical native northern adult is 11, and by a DEW Line worker is 0.9, based on the maximum concentrations of the COC (Tables 2-10 and A-11). The presence of barium, manganese, vanadium, and zinc accounts for the quantifiable noncancer hazard for these receptor/pathway combinations.

The excess lifetime cancer risk associated with the potential future ingestion of surface water at the site by a hypothetical native northern adult is 2×10^{-6}, and by a DEW Line worker is 2×10^{-8}, based on the maximum concentrations of the COC (Tables 2-10 and A-12). The presence of 1,2-dichloroethane entirely accounts for the quantifiable excess lifetime cancer risk for these receptor/pathway combinations.

2.5 RISK CHARACTERIZATION UNCERTAINTY

Several sources of uncertainty affect the estimates of excess lifetime cancer risk and noncancer hazard as presented in this risk assessment. The sources are generally associated with:

- Sampling and analysis of soil, sediment, and surface water;
- Assigning the source of contamination;
- Exposure assumptions, including estimates of exposure point concentrations;
- Evaluation of the toxicity of the COCs; and
- Methods and assumptions used to characterize the cancer risk and noncancer hazard.

Uncertainties associated with sampling and analysis include the inherent variability (standard error) in the analyses, representativeness of the samples, sampling errors, and heterogeneity of the sample matrix. The quality assurance/quality control program used in conducting the sampling and analyses serves to reduce errors, but it can not eliminate all errors associated with sampling and analyses. There is some uncertainty in the selection of COCs with respect to sample quantitation limits for a given chemical. In some cases a chemical may have had detected values below the COC screening criteria as well as samples with quantitation limits greater than the screening criteria. In these cases it should be understood that only the samples with adequate quantitation limits are applicable to the screening process. Thus, the number of

samples used to screen a chemical would be less than the total number of analyses for that chemical.

Simplifying assumptions were made about the environmental fate and transport of site contamination, specifically, no contaminant loss or transformation has occurred or will occur. Thus, the data chosen to represent exposure point concentrations in the sample-by-sample risk calculations are an additional source of potential error.

The depth at which a soil sample was collected was not considered in the risk characterization, so exposure to subsurface contamination was considered to be equally likely as exposure to surface contamination. This approach would tend to overestimate the true risk.

The estimation of exposure requires many assumptions to describe potential exposure situations. There are uncertainties regarding the likelihood of exposure, frequency of contact with contaminated media, the concentration of contaminants at exposure points, and the time period of exposure. These tend to simplify and approximate actual site conditions. In general, these assumptions are intended to be conservative and yield an overestimate of the true risk or hazard.

The toxicological database is also a source of uncertainty. The EPA has outlined some of the sources of uncertainty in the database (EPA 1986a,b; 1989a). These sources include extrapolation between exposure routes, from high to low doses, and from animals to humans; species, gender, age, and strain differences in uptake, metabolism, organ distribution, and target site susceptibility; and human population variability with respect to diet, environment, activity patterns, and cultural factors. The toxicity factors from IRIS and HEAST, which are used to estimate the toxicity of the COCs, are developed using a highly conservative methodology and probably tend to overestimate the potential hazards to humans.

Use of the provisional RfDs and SFs for DRPH, GRPH, and RRPH are an additional source of uncertainty in the toxicity assessment and risk characterization. Although the provisional RfDs represent the best available numerical estimate of toxicity, there is a significant amount of uncertainty associated with their use at the Wainwright installation. The RfDs and SFs are based on studies in mice and rats by the inhalation route of exposure; whereas, in this risk assessment, exposure of humans by the ingestion route only is being evaluated. Furthermore, in the absence of more thorough studies to compare to toxicity of DRPH, GRPH, and RRPH to the toxicity of known refinery streams, it is not clear how well the provisional values represent the toxicity of diesel, gasoline, and residual oils in humans.

In the risk characterization, the assumption was made that the total risk of developing cancer from exposure to site contaminants is the sum of the risk attributed to each individual contaminant. Likewise, the potential for the development of noncancer adverse effects is the sum of the HQs estimated for exposure to each individual contaminant. This approach does not account for the possibility that chemicals act synergistically or antagonistically but probably results in an overestimate of the true risk.

In addition to the more general sources of uncertainty associated with risk assessment methodology, there are site-specific sources of uncertainty. Primarily, these sources are

associated with the lifestyle of the native northerners, the time spent on the six sites that were investigated during the RI, and specific exposure assumptions (soil ingestion rate, exposure frequency, and exposure duration).

Inhabitants of Wainwright may use the installation occasionally as an access route for recreation (riding motorized vehicles) or subsistence hunting. No studies have been conducted to measure the time these potential receptors spend on contaminated sites at the installation. Some of the sites with levels of contamination that exceed regulatory benchmarks are not likely to be accessed by this group. Therefore, the assumptions made regarding exposure frequency probably result in an overestimate of the true noncancer hazard and cancer risk.

Similarly, no studies have been conducted to measure the soil ingestion rate of potential receptors on the contaminated sites. Potentially, soil ingestion by the inhabitants of Kaktovik may be greater than the default rate of 100 mg/day for adults and 200 mg/day for children. Given the rugged, partially subsistence, lifestyle of this group, it is possible that they incidentally ingest soil at a higher rate than receptors of a similar age in the continental United States. The estimate of soil ingestion rate used in this risk assessment may over- or underestimate the true rate.

The maximum exposure duration assumed for native northerners, 55 years, is probably fairly accurate. The RME estimate for inhabitants of the continental United States is 30 years; however, native northerners are more likely to remain in their villages for a longer period. Although, the exposure duration of 55 years is an estimate, it is not expected to significantly over- or underestimate hazard or risk.

2.6 RISK ASSESSMENT SUMMARY AND CONCLUSIONS

The human health risks associated with exposure to potentially contaminated media (soil, sediment, or surface water) at six sites at the Wainwright radar installation were evaluated in this risk assessment. The risk assessment was developed under a three step process:

1) The maximum concentrations of the chemicals detected in each medium (soil, sediment, or surface water) were compared to background concentrations, RBSLs, and ARARs. Chemicals present at concentrations that exceeded their background concentration and either an RBSL or an ARAR were retained as COCs for the risk assessment.

2) In the risk characterization, the noncancer HQ, excess lifetime cancer risk, or both were calculated based on the maximum concentration of each COC and its associated toxicity value developed by EPA.

3) The HQs for each COC at a given site were summed, and the sum (called a hazard index) was compared to the regulatory benchmark for noncancer hazard: a hazard index of one. Sites where the hazard index exceeded one were considered to warrant either remediation or further discussion.

The cancer risks for each carcinogenic COC at a given site were also summed, and the sum (the total cancer risk) was compared to the regulatory benchmark for cancer risks: an excess lifetime cancer risk of 1×10^{-6} (one in one million). Sites where the total cancer risk exceeded 1×10^{-6} are considered to warrant either remediation or further discussion. Sites where the hazard index was less than one and sites where the total cancer risk was less than 1×10^{-6} are considered to warrant no further action.

Table 2-11 contains a summary of the noncancer hazard and excess lifetime cancer risk for each site and medium at the Wainwright installation that exceeds a regulatory benchmark of 1.0 for noncancer hazard index or 1×10^{-6} for excess lifetime cancer risk.

No Further Action. Two of the six sites are considered to warrant no further action based on step 1: no chemical detected at these sites was classified as a COC based on a comparison of the maximum concentration measured soil, sediment, and surface water to the background concentration and the RBSL or ARAR. These two sites are the Drum Storage Area (ST02) and Airstrip Diesel (SS08).

The Diesel Fuel Spills (SS04) is considered to warrant no further action based on the hazard index and the total cancer risk. The hazard index for the soil at the site was less than 0.08 and the total cancer risk was less than 1×10^{-7} under both exposure scenarios evaluated.

The soil and sediment at the Vehicle Storage Area (SS09) and the Landfill (LF05) are considered to warrant no further action. No COCs were identified in the soil at the Vehicle Storage Area (thus, the site soils were eliminated). The hazard index for the soil at the Landfill was less than 0.001, and the total cancer risk less than 1×10^{-7}, under both exposure scenarios.

Sites that Warrant Further Discussion. The presence of 1,2-dichloroethane in the surface water at the Landfill (LF05) and the Vehicle Storage Area (SS09) accounts entirely for the cancer risk at these locations. At the Garage (SS07), bis(2-ethylhexyl)phthalate and 1,2-dichloroethane account for the cancer risk.

Both 1,2-dichloroethane and bis(2-ethylhexyl)phthalate are common laboratory contaminants and, although blank contamination did not affect interpretation of the laboratory analytical results, it is possible that these two organic compounds did not originate from activities associated with the operation of this radar installation because both compounds were infrequently detected and only at low concentrations.

In addition to the cancer risk, the hazard index associated with surface water ingestion at the Vehicle Storage Area (SS09) exceeds one due to the presence of manganese. Vanadium and zinc are also present but together contribute less than one percent of the total noncancer hazard.

As noted in the exposure assessment section, however, surface water ingestion is only a potentially complete pathway under a future use scenario. The future use scenario would require retirement of the Wainwright installation, release of the installation for civilian use, and redevelopment as a residential area in which the surface water was used as the sole source of

TABLE 2-11. SUMMARY OF SITES WITH CONTAMINATION THAT EXCEEDS REGULATORY BENCHMARKS
[Noncancer Hazard Index > 1.0, Excess Lifetime Cancer Risk > 1×10^{-6}]

SITE	MEDIUM	NONCANCER CHEMICALS OF CONCERN	CARCINOGENIC CHEMICALS OF CONCERN	NONCANCER HAZARD INDEX			EXCESS LIFETIME CANCER RISK		
				DEW LINE WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN ADULT/CHILD	DEW LINE WORKER	NATIVE NORTHERN ADULT	NATIVE NORTHERN ADULT/CHILD
Landfill (LF05)	Surface Water	—	1,2-Dichloroethane	—	—	—	—	8×10^{-6}	—
Garage (SS07)	Soil	DRPH GRPH RRPH Tetrachloroethene	—	—	—	3	—	—	—
	Surface Water	—	Bis(2-ethylhexyl)phthalate 1,2-Dichloroethane	—	—	—	—	5×10^{-6}	—
Vehicle Storage Area (SS09)	Surface Water	Barium Manganese Vanadium Zinc	1,2-Dichloroethane	—	11	—	—	2×10^{-6}	—

[a] All COCs are listed regardless of whether they contribute to the hazard index or cancer risk.

water for domestic purposes. Such a scenario is not likely because the community of Wainwright and its existing water supply system already adequately support the native population in the area and there are no population growth pressures to force development of additional land or water resources. In addition, there is no road connecting the community of Wainwright with the Wainwright radar installation.

The hazard index associated with incidental soil ingestion at the Garage (SS07) exceeds one for the native northern adult/child receptor group, based on the presence of DRPH and RRPH. This pathway is only potentially complete and, like the surface water pathway, would require retirement of the Wainwright installation, release of the installation for civilian use, and redevelopment as a residential area. Such a scenario is not currently likely to occur because the Air Force is operating the Wainwright installation as an automated, unmanned radar installation.

In conclusion, under current uses the COCs at the Wainwright installation pose only minimal, if any, potential threat to human health. Based on the human health risk assessment, remedial actions/cleanups are not necessarily warranted at any of the six sites.

THIS PAGE INTENTIONALLY LEFT BLANK

3.0 ECOLOGICAL RISK ASSESSMENT

The objective of the ERA is to estimate potential impacts to aquatic and terrestrial plants and animals at the Wainwright DEW Line installation. This document assesses potential ecological risks at the Wainwright installation based on sampling and analyses conducted during the RI of the six installation sites. The RI was completed during the summer of 1993 in conjunction with RIs at seven other radar installations.

Guidance documents used during preparation of this assessment include:

- Handbook to Support the Installation Restoration Program Statements of Work (U.S. Air Force 1991);
- Framework for Ecological Risk Assessment (EPA 1992a); and
- Ecological Risk Assessment Guidance for Superfund (EPA 1994).

The approach used to assess potential ecological impacts is conceptually similar to that for human health risks; potentially exposed populations (receptors) are identified, and then information on exposure and toxicity are combined to derive estimates of risk. The ecological assessment focuses, however, on potential impacts to a population of organisms rather than to individual organisms (except in the case of endangered species where individuals are considered). Because ecosystems are composed of a variety of species, ecological assessments evaluate potential impacts to numerous species.

Ideally, ERAs should evaluate potential risks to communities and ecosystems, as well as to individual populations. Because of the large number of species and communities present in natural systems such ecosystem-wide assessments are very complex and appropriate assessment methodologies have not yet been developed. In addition, dose-response data on community or ecosystem responses are generally lacking. Therefore, evaluations of potential impacts to communities or ecosystems are qualitative.

The degree to which potential ecological impacts can be characterized is highly dependent upon the data available to support such estimates. Required data are those regarding contaminant release, transport, and fate; characteristics of potential receptor populations; and toxicity of the chemicals evaluated.

This ERA is intended to be at a screening level, rather than a full scale investigation of the state of the ecosystem. No site-specific studies of the biota were undertaken. The assessment is based on media sampling (i.e., surface water and soil/sediment samples). It is divided into six sections:

Section 3.1 - Selection of Site Contaminants;
Section 3.2 - Exposure Assessment;
Section 3.3 - Ecological Toxicity Assessment;

Section 3.4 - Risk Characterization for Ecological Receptors;
Section 3.5 - Ecological Risk Assessment Uncertainty Analysis; and
Section 3.6 - Summary of Ecological Risk.

3.1 SELECTION OF SITE CONTAMINANTS

A stressor in the environment is a chemical, physical, or biological action that can cause a negative impact on an ecosystem (EPA 1992b). Only chemical stressors identified as chemicals of concern (COCs) are evaluated as part of this ERA. A review of the site data indicates that the chemical stressors are primarily petroleum products, solvents, and metals.

The six sites at the Wainwright DEW Line installation are all considered to be potentially suitable habitat for ecological receptors because the installation has been inactive since 1989. As a result, human activities that would discourage use by representative species are limited and not expected to deter species from frequenting the installation.

COCs are selected based on comparisons to background concentrations and action levels[1]. If no action levels were available, the maximum detected concentration of the chemical was compared to a toxicity value derived from acute or chronic exposure tests available in the literature. If the maximum concentration was above this level, the compound was considered a COC. Chemicals present onsite at concentrations in excess of background concentrations and action levels were evaluated for frequency of detection in onsite media. If a chemical was detected at a frequency of less than 5 percent, it was not considered representative of actual site conditions and was eliminated from evaluation in the risk assessment. Further, an attempt was made to eliminate elements that were within the range of natural background levels. To that end, if the average concentration (exposure concentration) of a chemical was lower than the maximum background concentration (i.e., if the average falls within the range of background), and if the maximum detected concentration was less than twice the maximum background concentration[2], the chemical was not considered a COC. Tables 3-1 and 3-2 present the data used in the screening process for surface water and soil/sediment. Only chemicals that were detected in at least one environmental sample are presented in these summary tables.

---

1 Action Levels: Federal Ambient Water Quality Criteria for surface water (AWQC); ADEC Water Quality Standards (18 AAC 70.020[b]) January 1995; Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota (Suter and Mabrey 1994); ADEC determination of cleanup levels for petroleum contaminated soils (Interim Guidance for Non-UST Contaminated Soil Cleanup Levels, 17 July 1991); EPA sediment quality criteria (as estimated by Hull and Suter 1994); and, NOAA Sediment Effects Range (Low) (NOAA 1991).

2 This is meant to approximate the 95 percent upper confidence limit (UCL) of background concentrations.

TABLE 3-1. SUMMARY OF CHEMICALS OF CONCERN: SURFACE WATER

CHEMICALS OF CONCERN: WAINWRIGHT INSTALLATION SURFACE WATER						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (µg/L)	BACKGROUND RANGE FROM 7 DEW LINE INSTALLATIONS (µg/L)	ACTION LEVEL (µg/L)	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (µg/L)	SELECTED AS COC
ORGANICS						
1,2-Dichloroethane	1.6 - 6.2	<1	20,000[a]	3/8	1.5	NO
Bis(2-ethylhexyl)phthalate	16	<10 - <13	32.2[b]	1/8	7	NO
INORGANICS - based on total metals						
Aluminum	180 - 9,700	<100 - 350	87[c]	3/6	2,000	**YES**
Barium	53 - 750	<50 - 93	5,800[d]	6/6	280	NO
Calcium	6,000 - 71,000	4,500 - 88,000	116,000[d]	6/6	35,000	NO
Iron	1,100 - 130,000	180 - 2,800	1,000[c]	6/6	28,000	**YES**
Magnesium	6,000 - 62,000	<5,000 - 53,000	82,000[d]	6/6	40,000	NO
Manganese	130 - 3,800	<50 - 510	80.3[b]	3/6	690	**YES**
Nickel	51	<50	160[c,e]	1/6	29	NO
Potassium	9,500	<5,000	53,000[d]	1/6	3,700	NO
Sodium	18,000 - 110,000	8,400 - 410,000	680,000[d]	6/6	50,000	NO
Vanadium	63	<50	19.1[d]	1/6	31	**YES**
Zinc	230 - 3,300	<50 - 160	110[c,e]	3/6	670	**YES**

a EPA AWQC: insufficient data to develop criteria. Value presented in the Lowest Observed Effect Level (LOEL).
b Based on the Secondary Chronic Value presented in Suter and Mabrey (1994), following methods described in the EPA's *Proposed water quality Guidance for the Great Lakes System* (used if AWQC not available).
c EPA AWQC, Fresh water chronic criteria.
d Based on the Lowest Chronic Value for All Organisms presented in Suter and Mabrey (1994) (used if AWQC not available).
e Hardness dependent criteria (100 mg/L $CaCO_3$ used). Site-specific hardness not available.

TABLE 3-2. SUMMARY OF CHEMICALS OF CONCERN: SOILS AND SEDIMENTS

CHEMICALS OF CONCERN: WAINWRIGHT INSTALLATION SEDIMENT AND SOIL						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (mg/kg)	BACKGROUND RANGE (mg/kg)	ACTION LEVEL (mg/kg)[a]	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (mg/kg)	SELECTED AS COC
ORGANICS						
DRPH	47.4 - 120,000	<50 - <300	500[b]	8/43	3,190	**YES**
GRPH	6NJ - 200J	<2J - <5J	100[b]	7/41	14	**YES**
RRPH	360 - 77,000	<100 - <600	2,000[b]	4/43	1,900	**YES**
Toluene	0.172 - 0.205	<0.02 - <0.1	0.786	2/39	0.1	NO
Ethylbenzene	0.1NJ - 6.5NJ	<0.02 - <0.1	4.36[c]	4/39	0.32	**YES**
Xylenes (Total)	0.022 - 17NJ	<0.04 - <0.2	1.21	6/39	1.0	**YES**
Tetrachloroethene	0.059 - 10.9	<0.020 - <0.5	2.73[c]	4/29	1.1	**YES**
p-Isopropyltoluene	0.169 - 0.448	<0.020 - <0.400	4.36[d]	2/11	0.1	NO
Naphthalene	0.034 - 0.732	<0.020 - <0.400	0.34	3/11	0.1	**YES**
1,1,1-Trichloroethane	0.062	<0.020 - <0.400	0.179[c]	1/11	0.1	NO
1,2,4-Trimethylbenzene	0.040 - 0.616	<0.020 - <0.400	--	3/11	0.1	**YES**
1,3,5-Trimethylbenzene	0.024 - 9.95	<0.020 - <0.400	--	4/11	1.3	**YES**
Di-n-Butylphthalate	1.6 B - 37.6BJ	1.69 U - 83.4J	42.1[c]	3/5	10	NO

-- Not available.
a NOAA 1991, sediment ER-L (Effects Range - low).
b ADEC, Interim Guidance for Non-UST Contaminated Soil Cleanup Levels 17 July 1991.
c EPA Sediment Quality Criteria (estimated using equilibrium partitioning approach summarized in Hull and Suter 1994).
d EPA Sediment Quality Criteria for Ethylbenzene (see text).
e Hardness dependent criteria (100 mg/L $CaCO_3$ used). Site-specific hardness not available.
B Detected in blanks.
NJ Estimated value, presumptive evidence.
U Compound not preset above concentrations listed.

TABLE 3-2. SUMMARY OF CHEMICALS OF CONCERN: SOILS AND SEDIMENTS (CONTINUED)

CHEMICALS OF CONCERN: WAINWRIGHT INSTALLATION SEDIMENT AND SOIL						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (mg/kg)	BACKGROUND RANGE (mg/kg)	ACTION LEVEL (mg/kg)[a]	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (mg/kg)	SELECTED AS COC
INORGANICS						
Aluminum	1,200 -15,000	1,500 - 25,000	--	8/8	4,500	NO
Barium	59 - 420	27 - 390	--	8/8	210	NO
Cadmium	72	<3.0 - <36	5[a]	1/8	19	**YES**
Calcium	910- 15,000	360 - 59,000	--	8/8	4,500	NO
Chromium	5.3 - 26	4.3 - 47	80	4/8	14	NO
Cobalt	3.8 - 15	<5.1 - 12	--	4/8	7	NO
Copper	4.8 - 41	<2.7 - 45	70	8/8	16	NO
Iron	12,300 - 114,000	5,400 - 35,000	--	8/8	57,000	**YES**
Lead	19 - 102	<5.1 - 22	35	3/8	30	**YES**
Magnesium	800 - 5,300	360 - 7,400	--	8/8	2,400	NO
Manganese	67 - 1,400	25 - 290	--	8/8	610	**YES**
Nickel	5.5 - 29	4.2 - 46	30	8/8	17	NO
Potassium	230 - 1,500	<300 - 2,200	--	8/8	600	NO

-- Not available.
a NOAA 1991, sediment ER-L (Effects Range - low).
b ADEC, Interim Guidance for Non-UST Contaminated Soil Cleanup Levels 17 July 1991.
c EPA Sediment Quality Criteria (estimated using equilibrium partitioning approach summarized in Hull and Suter 1994).
d EPA Sediment Quality Criteria for Ethylbenzene (see text).
e Hardness dependent criteria (100 mg/L $CaCO_3$ used). Site-specific hardness not available.
B Detected in blanks.
NJ Estimated value, presumptive evidence.
U Compound not preset above concentrations listed.

TABLE 3-2. SUMMARY OF CHEMICALS OF CONCERN: SOILS AND SEDIMENTS (CONTINUED)

CHEMICALS OF CONCERN: WAINWRIGHT INSTALLATION SEDIMENT AND SOIL						
CHEMICAL	RANGE OF DETECTED CONCENTRATIONS (mg/kg)	BACKGROUND RANGE (mg/kg)	ACTION LEVEL (mg/kg)[a]	FREQUENCY OF DETECTION	AVERAGE CONCENTRATION FOR COC (mg/kg)	SELECTED AS COC
INORGANICS (CONTINUED)						
Sodium	52 - 1,500	<160 - 680	--	8/8	380	NO
Vanadium	9 - 43	6.3 - 59	--	8/8	28	NO
Zinc	23 - 240	9.2 - 95	120	8/8	81	**YES**

-- Not available.
a NOAA 1991, sediment ER-L (Effects Range - low).
b ADEC, Interim Guidance for Non-UST Contaminated Soil Cleanup Levels 17 July 1991.
c EPA Sediment Quality Criteria (estimated using equilibrium partitioning approach summarized in Hull and Suter 1994).
d EPA Sediment Quality Criteria for Ethylbenzene (see text).
e Hardness dependent criteria (100 mg/L $CaCO_3$ used). Site-specific hardness not available.
B Detected in blanks.
NJ Estimated value, presumptive evidence.
U Compound not preset above concentrations listed.

In summary, the decisions for selecting COCs were made using the following logic:

STEP ONE: Is the chemical detected above the maximum detected background concentration?

No: Not considered a COC.
Yes: Continue to step two.

STEP TWO: Is the chemical detected above the action level or toxicity value?

No: Not considered a COC.
Yes: Continue to step three.

STEP THREE: Is the chemical detected at a frequency greater than five percent?

No: Not considered a COC.
Yes: Continue to step four.

STEP FOUR: Is the average concentration of the chemical greater than the maximum background concentration and is the maximum detected concentration greater than twice the maximum background concentration?

No: Not considered a COC.
Yes: Chemical is classified as a COC.

All data for COCs were averaged (arithmetic mean) according to media. In the case of non-detects, averages were calculated using one-half of the quantitation limits. Replicate samples were averaged and treated as one sample. Total metal concentrations were used in determining COCs in surface water. This is a conservative approach because dissolved metal concentrations (the more bioavailable fraction) can be significantly lower than total metal concentrations. Section 3.1.1 describes surface water COCs. Section 3.1.2 describes soil and sediment COCs.

3.1.1 Surface Water

Analytical results from the six sites were compiled and evaluated to determine the COCs. Surface water samples were collected and analyzed for contaminants likely to be present at the specific sites. Not all samples were analyzed for a "full suite" of parameters, but instead were analyzed for some combination of the following: DRPH, GRPH, RRPH, benzene, toluene, ethylbenzene, and xylene (BTEX), halogenated volatile organic compounds (HVOCs), VOCs, SVOCs, polychlorinated biphenyls (PCBs), pesticides, and metals (all metals reported are total metals unless noted otherwise). A summary of analytical results for all sampling conducted at the installation is presented in Appendix G. This section presents the evaluation of the surface water data. Table 3-1 summarizes the screening and selection of COCs in surface water.

3.1.1.1 Organic Compounds. Two organic compounds were detected in surface water samples collected from Wainwright installation: 1,2-dichloroethane and bis(2-ethylhexyl)phthalate. This section presents the evaluation of these compounds as COCs in surface water for the ERA.

1,2-Dichloroethane was detected in three of eight surface water samples collected at the Wainwright facility and analyzed for VOCs. Concentrations ranged from 1.6 to 6.2 µg/L. 1,2-dichloroethane was not detected in background samples above the detection limit of 1 µg/L. The action level of this compound is 20,000 µg/L, based on ambient water quality criterion (AWQC) lowest observed effect level (LOEL). Because 1,2-dichloroethane was detected at levels substantially below the action level this compound is not considered a COC.

Bis(2-ethylhexyl)phthalate was detected in one of eight surface water samples analyzed for SVOCs at a concentration of 16 µg/L. The AWQC for this compound in 1992 was based on the group of phthalate esters and was reported as 3 µg/L. This value is currently under revision by the EPA (Charles Delos, pers. comm. 1995). As a result, the action level used in this assessment is based on *Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision* (Suter and Mabrey 1994). Suter and Mabrey (1994) report a Tier II Secondary Chronic Value of 32.2 µg/L for bis(2-ethylhexyl)phthalate. This value is calculated based on a method described in the EPA's *Proposed Water Quality Guidance for the Great Lakes System* (Suter and Mabrey 1994). The calculation incorporates the following acute toxicity values: 2,000 µg/L (*Daphnia magna*); 11,000 µg/L (*Daphnia magna*); 133 µg/L (*Daphnia pulex*); >100,000 µg/L (Channel catfish); >100,000 µg/L (coho salmon); >100,000 µg/L (bluegill); and >32,000 µg/L (scud). These values indicate the range of concentrations that may be expected to elicit acute effects to aquatic organisms and demonstrate that this compound is not extremely toxic to aquatic life. As a result, a chronic value of 32.2 appears to be a suitable screening level value. Bis(2-ethylhexyl)phthalate was detected in one out of eight samples, but at a concentration below the action level, so it is not considered a COC.

3.1.1.2 Metals. Six surface water samples collected from three of the six sites at the Wainwright installation were analyzed for metals. The sites where samples for metal analyses were collected are: LF05 (Landfill); SS07 (Garage); and SS09 (Vehicle Storage Area). Eleven inorganic analytes were detected: aluminum, barium, calcium, iron, magnesium, manganese, nickel, potassium, sodium, vanadium, and zinc. This section presents the evaluation of these metals as COCs for the ERA. Analytes not detected in surface water samples were: antimony, arsenic, beryllium, cadmium, chromium, cobalt, copper, lead, molybdenum, selenium, silver, and thallium. It is important to note that in some cases, detection limits for certain metals were somewhat higher than ecologically relevant action levels. For example, in the case of copper, the detection limit was 50 µg/L. However, the current AWQC (based on a hardness value of 100 mg/L $CaCO_3$) for copper is 12 µg/L. As a result, an ecological risk may exist for aquatic organisms from exposure to certain metals at their detection limits. These metals include: cadmium, chromium (VI), copper, lead, and selenium. These issues will be further addressed in Section 3.5, Uncertainty Analysis.

Aluminum was detected in three of six surface water samples. Concentrations ranged from 180 to 9,700 µg/L. Background concentrations ranged from <100 to 350 µg/L. The EPA chronic AWQC for aluminum is 87 µg/L; this value was used as the action level. Aluminum is present in

surface water at concentrations in excess of the action level, so it was retained as a COC. The exposure concentration evaluated in this ERA is the average concentration of 2,000 μg/L.

Barium was detected in all six surface water samples. Concentrations ranged from 53 to 750 μg/L. Background concentrations of barium ranged from <50 to 93 μg/L. The action level for barium is 5,800 μg/L, based on data presented in Suter and Mabrey (1994). The value selected as the action level is the Lowest Chronic Value for All Organisms. Although barium concentrations exceed background levels, this chemical was not retained as a COC because it does not exceed the action level.

Calcium was detected in all six surface water samples. Concentrations ranged from 6,000 to 71,000 μg/L. Background concentrations ranged from 4,500 to 88,000 μg/L. The action level is 116,000 μg/L based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994). Calcium does not exceed background concentrations or the action level, so it was not retained as a COC.

Iron was detected in all six surface water samples. Concentrations ranged from 1,100 to 130,000 μg/L. Background concentrations ranged from 180 to 2,800 μg/L. Iron exceeds the background concentration in surface water and the 1,000 μg/L action level based on the AWQC, so this metal was retained as a COC. The exposure concentration evaluated in this ERA is the average concentration of 28,000 μg/L.

Magnesium was detected in all six surface water samples. Concentrations ranged from 6,000 to 62,000 μg/L. Background concentrations ranged from <5,000 to 53,000 μg/L. There is no AWQC for magnesium. An action level of 82,000 μg/L was used based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994). Magnesium was not retained as a COC in the ERA because it is present onsite at concentrations closely approximating background concentrations that do not exceed the action level.

Manganese was detected in three out of six surface water samples. Concentrations ranged from 130 to 3,800 μg/L. Background concentrations ranged from <50 to 510 μg/L. The action level for manganese is 80.3 μg/L based on the Secondary Chronic Value presented in Suter and Mabrey (1994). Because manganese was detected at concentrations in excess of background and action levels, it was retained as a COC. The exposure concentration evaluated in this ERA is the average concentration of 690 μg/L.

Nickel was detected in one out of six surface water samples at a concentration of 51 μg/L. Nickel was not detected in background samples above the detection limit of 50 μg/L. The chronic AWQC for nickel is 160 μg/L based on a hardness of 100 mg/L $CaCO_3$. Because nickel was not detected in excess of action levels it was not retained as a COC.

Potassium was detected in one of six surface water samples at a concentration of 9,500 μg/L. This element was not detected in background samples above the detection limit of 5,000 μg/L. The action level for potassium is based on the Lowest Chronic Value for All Organisms of 53,000 μg/L (Suter and Mabrey 1994). Because concentrations of potassium did not exceed the action level, it was not selected as a COC.

Sodium was detected in all six surface water samples. Concentrations ranged from 18,000 to 110,000 µg/L, below the background concentrations of 8,400 to 410,000 µg/L. The action level for sodium of 680,000 µg/L was based on the Lowest Chronic Value for All Organisms (Suter and Mabrey 1994). Onsite concentrations did not exceed background levels or the action level, so sodium was not selected as a COC.

Vanadium was detected in one out of six samples at a concentration of 63 µg/L. Vanadium was not detected in the background samples at <50 µg/L. The action level for vanadium is based on the Secondary Chronic Value of 19.1 µg/L (Suter and Mabrey 1994). Vanadium was detected above action levels, so it was retained as a COC. Vanadium will be evaluated in the risk assessment. The average concentration evaluated in this risk assessment is 31 µg/L.

Zinc was detected in 3 out of 6 surface water samples at concentrations ranging from 230 to 3,300 µg/L. Background concentrations of zinc ranged from <50 - 160 µg/L. The chronic AWQC for zinc is 110 µg/L based on a water hardness of 100 mg/L $CaCO_3$. Because concentrations of zinc in surface water exceeded background concentrations and chronic AWQC, zinc was retained as a COC in the risk assessment. The average concentration evaluated in the risk assessment is 670 µg/L.

3.1.2 Soils and Sediments

Soil/sediment sample analytical results from the six sites were compiled and evaluated to determine the COCs. Samples were collected and analyzed for contaminants likely to be present at the specific sites. Not all samples were analyzed for a "full suite" of parameters, but instead were analyzed for some combination of the following: DRPH, GRPH, RRPH, BTEX, HVOCs, VOCs, SVOCs, PCBs, pesticides, and metals. A summary of analytical results for all sampling conducted at the installation is presented in Appendix G. This section presents the evaluation of the soil/sediment data for the six sites. Only compounds that were detected on the site are discussed. Table 3-2 summarizes the screening results.

3.1.2.1 Petroleum Hydrocarbons. Forty-three soil/sediment samples were collected from the six sites and selectively analyzed for a combination of DRPH and RRPH. Forty-one soil/sediment samples were collected and analyzed for GRPH. A discussion of these petroleum hydrocarbon mixtures and their toxicity is presented in Section 3.3.1.

Diesel Range Petroleum Hydrocarbons were detected in 8 of 43 soil/sediment samples at concentrations ranging from 47.4 to 120,000 mg/kg; background concentrations ranged from <50 to <300 mg/kg. The action level for DRPH in soils/sediments is 500 mg/kg. Because DRPH were detected at levels above the action level, they were retained as a COC. The exposure concentration used in the risk assessment is the average concentration of 3,190 mg/kg.

Gasoline Range Petroleum Hydrocarbons were detected in 7 of 41 soil/sediment samples ranging from 6 to 200 mg/kg. The background concentrations ranged from <2 to <5 mg/kg. The action level for GRPH is 100 mg/kg. GRPH were detected at concentrations above the action level, so they are considered a COC. The exposure concentration used in this assessment is the average of 14 mg/kg.

Residual Range Petroleum Hydrocarbons were detected in 4 of 43 samples ranging from 360 to 77,000 mg/kg. The background concentrations for RRPH ranged from <100 to <600 mg/kg; the action level is 2,000 mg/kg. RRPH were detected at concentrations above the action level, so they are considered a COC. The average concentration is 1,900 mg/kg.

3.1.2.2 Benzene, Toluene, Ethylbenzene, and Xylenes. Thirty-nine soil/sediment samples were collected from the six sites at the Wainwright installation and analyzed for BTEX by the 8020/8020 modified method. In addition, toluene and xylenes were detected using the VOC (8260) analysis. In the case of these duplicate analyses, the average concentration was calculated from the analytical method that produced the highest concentration. This is a conservative approach that is expected to be protective of ecological receptors. The following paragraphs summarize the analytical results.

Toluene was detected in 2 of 39 soil/sediment samples at concentration of 0.172 and 0.205 mg/kg. The background concentration ranged from <0.02 to <0.1 mg/kg. The action level for this compound is 0.786 mg/kg. Because concentrations at the site did not exceed the action level, toluene was not retained as a COC.

Ethylbenzene was detected in 4 of 39 soil/sediment samples at concentrations ranging from 0.1 to 6.5 mg/kg. The background concentration of ethylbenzene ranged from <0.02 to <0.1 mg/kg for soil/sediment; the action level is 4.36 mg/kg. Onsite concentrations exceed the action level, so ethylbenzene is considered a COC. The exposure concentration used in this ERA is the average concentration of 0.32 mg/kg.

Xylene was detected in 6 of 39 samples. Xylene concentrations ranged from 0.022 to 17 mg/kg. The background concentration of xylene ranged from <0.04 to <0.2 mg/kg. The action level is 1.21 mg/kg. Xylene is considered a COC as onsite concentrations are above the action level. The exposure concentration used in this ERA is the average concentration of 1.0 mg/kg.

3.1.2.3 Halogenated Volatile Organic Compounds and Volatile Organic Compounds. Six HVOCs/VOCs were detected in soil/sediment samples collected from Wainwright. The compounds detected were tetrachloroethene, p-isopropyltoluene, naphthalene, 1,1,1-trichloroethane, 1,2,4-trimethylbenzene, and 1,3,5-trimethylbenzene. This section presents the evaluation of these compounds as COCs for the ERA.

Tetrachloroethene was detected in 4 of 29 soil/sediment samples at concentrations ranging from 0.059 to 10.9 mg/kg. The background concentration ranges from <0.020 to <0.5 mg/kg. The action level for this compound is 2.73 mg/kg (Hull & Suter 1994). Because this compound was detected at high frequency at concentrations above the action level, it is considered a COC. The exposure concentration used in this assessment is the average concentration of 1.1 mg/kg[3].

p-Isopropyltoluene was detected in two out of eleven soil/sediment samples at concentrations of 0.169 and 0.448 mg/kg. This chemical was not detected in background samples at detection

---

3 Tetrachloroethene was detected under the analytical methods for HVOC and VOCs. The analytical method that produced the highest average concentration (VOC) was used for the exposure concentration.

limits of <0.020 to <0.400 mg/kg. There are no action levels for this compound; however, an action level for a similar compound, ethylbenzene, was used. This action level is 4.36 mg/kg. Because this compound was detected below the action level, it was not retained as a COC.

Naphthalene was detected in three of eleven soil/sediment samples at concentrations ranging from 0.034 to 0.732 mg/kg. This chemical was not detected in background samples at detection limits of <0.020 to <0.400 mg/kg. The action level for this compound is 0.34 mg/kg. It was retained as a COC, and the exposure concentration evaluated in this ERA is the average concentration of 0.1 mg/kg.

1,1,1-Trichloroethane was detected in one of eleven soil/sediment samples. The detected concentration was 0.062 mg/kg. The action level for this compound is 0.179 mg/kg. This compound was not detected in background samples (<0.020 - <0.400 mg/kg). Because this compound was not detected above the action level, it was not retained as a COC.

1,2,4-Trimethylbenzene was detected in three of eleven samples at concentrations ranging from 0.040 to 0.616 mg/kg. This compound was not detected in background samples at detection limits of <0.020 to <0.400. There is no action level for this compound. Because this compound was detected at high frequency (albeit at low concentrations), this chemical was retained as a COC. The concentration evaluated in this assessment is 1.4 mg/kg (see discussion of 1,3,5-trimethylbenzene).

1,3,5-Trimethylbenzene was detected in four of eleven samples at concentrations ranging from 0.024 to 9.95 mg/kg. This compound was not detected in background samples at detection limits of <0.020 to <0.400 mg/kg. There is no action level for this compound. This compound is considered a COC, with its related isomer 1,2,4-trimethylbenzene. Because of the lack of toxicological data for these isomers, these compounds will be considered the same, and the sum of the average concentrations for both isomers will be used as the exposure concentration for both (1.4 mg/kg).

3.1.2.4 Semivolatile Organic Compounds. Di-n-Butylphthalate was the only SVOC detected in soil/sediment samples from the Wainwright installation.

di-n-Butylphthalate was detected in three out of five soil/sediment samples at concentrations ranging from 1.6 to 37.6 mg/kg. This chemical is a common laboratory contaminant (EPA 1989) and was detected in the blank samples associated with these results. di-n-Butylphthalate was not considered a COC in the risk assessment because the detected concentrations did not exceed the action level of 42.1 mg/kg (Hull and Suter 1994).

3.1.2.5 Metals. Sixteen inorganic analytes were detected in eight soil/sediment samples collected from the Wainwright installation. The metals detected were aluminum, barium, cadmium, calcium, chromium, cobalt, copper, iron, lead, magnesium, manganese, nickel, potassium, sodium, vanadium, and zinc. This section presents the evaluation of these metals as COCs for the ERA.

Aluminum was detected in all eight soil/sediment samples. Concentrations ranged from 1,200 to 15,000 mg/kg. Background concentrations ranged from 1,500 to 25,000 mg/kg. There is no action level for aluminum. Onsite concentrations did not exceed background concentrations, so aluminum was not retained as a COC.

Barium was detected in all eight soil/sediment samples at concentrations between 59 and 420 mg/kg. The background concentrations of barium ranged from 27 to 390 mg/kg. The average concentration of barium was 210 mg/kg. There is no action level for barium. Because the last selection criteria for COCs is not met (i.e., the average concentration of barium is less than the maximum background concentration and the maximum detected concentration is less than twice the maximum background concentration), levels of barium are not considered elevated above background conditions. As a result, barium was not retained as a COC.

Cadmium was detected in one of eight soil/sediment samples at 72 mg/kg. Cadmium was not detected in background samples (<3.0 - <36 mg/kg). The action level for cadmium is 5 mg/kg. Because cadmium was detected at concentrations above action levels, it was retained as a COC. The average concentration used in this assessment is 19 mg/kg.

Calcium was detected in all eight soil/sediment samples. Concentrations ranged from 910 to 15,000 mg/kg. Background concentrations ranged from 360 to 59,000 mg/kg. There is no action level for calcium. Onsite concentrations did not exceed background concentrations, so this chemical was not retained as a COC.

Chromium was detected in four of eight soil/sediment samples. Concentrations ranged from 5.3 to 26 mg/kg. The maximum background concentration is 47 mg/kg. The action level for chromium is 80 mg/kg. The detected concentrations did not exceed the action level or background concentrations, so this metal was not retained as a COC.

Cobalt was detected in four of eight samples collected from soil/sediment at the Wainwright installation. Concentrations ranged from 3.8 to 15 mg/kg and were in excess of the background range of <5.1 to 12 mg/kg. There is no action level for cobalt. The average onsite concentration is 7 mg/kg. Because the average concentration of cobalt is less than the maximum background concentration and the maximum detected concentration is less than twice the maximum background concentration, cobalt was not retained as a COC.

Copper was detected in all soil/sediment samples collected from the Wainwright installation. Detected concentrations ranged from 4.8 to 41 mg/kg. Background concentrations ranged from <2.7 to 45 mg/kg. The action level for copper is 70 mg/kg. Because the copper was not detected above the maximum detected background concentration copper was not retained as a COC.

Iron was detected in all eight soil/sediment samples. Concentrations ranged from 12,300 to 114,000 mg/kg. The background concentrations ranged from 5,400 to 35,000 mg/kg. There is no action level for iron. This metal was retained as a COC because onsite concentrations exceeded background concentrations. The exposure concentration evaluated for iron in this ERA is the average concentration of 57,000 mg/kg.

Lead was detected in three of eight soil/sediment samples. Concentrations ranged from 19 to 102 mg/kg. The maximum background concentration for lead is 22 mg/kg. The action level for lead is 35 mg/kg. Lead concentrations exceeded background and action levels; therefore, lead was retained as a COC. The exposure concentration evaluated in this ERA is the average concentration of 30 mg/kg.

Magnesium was detected in all eight soil/sediment samples. Concentrations ranged from 800 to 5,300 mg/kg. The background concentrations for magnesium ranged from 360 to 7,400 mg/kg. There is no action level for magnesium. Because magnesium was not detected above background concentrations, it was not retained as a COC.

Manganese was detected in all eight soil/sediment samples. Concentrations ranged from 67 to 1,400 mg/kg. The background concentrations for manganese ranged from 25 to 290 mg/kg. There are no action levels for manganese. Because onsite concentrations exceeded background concentrations, this chemical was retained as a COC. The average concentration that will be evaluated in the risk assessment is 610 mg/kg.

Nickel was detected in all eight soil/sediment samples ranging in concentration from 5.5 to 29 mg/kg. The background concentrations ranged from 4.2 to 46 mg/kg. The action level for nickel is 30 mg/kg. This metal was not retained as a COC because onsite concentrations did not exceed background concentrations or the action level.

Potassium was detected in all eight soil/sediment samples. Concentrations ranged from 230 to 1,500 mg/kg. The background concentrations ranged from <300 to 2,200 mg/kg. There is no action level for potassium. This metal was not retained as a COC because onsite concentrations were below background concentrations.

Sodium was detected in all eight soil/sediment samples. Concentrations detected ranged from 53 to 1,500 mg/kg, which exceeded the maximum background concentration of 680 mg/kg. There is no action level for sodium. The average concentration of sodium is 380 mg/kg. Although the maximum detected sodium concentration exceeded twice the maximum background concentration , 1,360 mg/kg, it was not retained as a COC because the average reference level for sodium in soils is 5,300 mg/kg based on Lindsay (1979), which is above the detected concentrations at the Wainwright facility. Further, sodium is ubiquitous in the environment, is considered essential in the diet of mammals and is not expected to pose a threat to ecological receptors.

Vanadium was detected in every soil/sediment sample ranging in concentration from 9 to 43 mg/kg. The background concentrations ranged from 6.3 to 59 mg/kg. There is no action level for vanadium. This metal was not retained as a COC because onsite concentrations were below background concentrations.

Zinc was detected in every soil/sediment sample at concentrations from 23 to 240 mg/kg. The background concentrations for zinc ranged from 9.2 to 95 mg/kg. The action level for zinc is 120 mg/kg. Zinc was detected in excess of the action level, so zinc was retained as a COC. The exposure concentration evaluated in this ERA is the average concentration of 81 mg/kg.

3.2 ECOLOGICAL EXPOSURE ASSESSMENT

The vegetation of the Arctic Coastal Plain and the ecosystems it characterizes have developed primarily as a result of the low relief and harsh environment. The growing season is short, typically extending from June through mid-September. Winters are long, cold, dry, and dark. Air temperatures that average below freezing for most of the year result in a permafrost layer that begins near the surface and reaches to depths as great as 610 meters. Seasonal thawing results in an active layer between ground surface and 3.7 meters below the surface (Hart Crowser 1987).

The impervious permafrost layer prevents percolation and infiltration of water below the active layer, and the generally flat terrain provides poor drainage. As a result, the ecosystems of the Arctic Coastal Plain are often defined not only by their plant associations but also by the degree of water found in and on them. Hart Crowser (1987) describes five major ecosystems for the classification of tundra and Arctic Coastal Plain communities:

- <u>Marine zones</u>: these include lagoons, estuaries, barrier islands, strands, and beaches. The abundance of vegetation along the marine coastal zone is inversely related to the amount of beach scouring by waves and ice. Mainland beaches support a variety of vegetation, including sedges, grasses, and forbs.

- <u>Wet sedge meadows</u>: an association of meadows, ponds, and lakes also known as "wet tundra". This system, with its associated wetlands, is dominant in the area extending west from the Colville River to the Chukchi Sea (including the Point Lonely, Point Barrow, Wainwright, Point Lay, and Cape Lisburne installations). Differences in vegetation within this ecosystem are related to moisture and microrelief.

- <u>Tussock tundra</u>: or "moist tundra" consisting primarily of areas dominated by tussock-forming cottongrass. This system covers significant portions of the Arctic Coastal Plain.

- <u>Riverine systems and floodplains</u>: including riparian shrubland on recent and old alluvium. Being better drained than surrounding lands, the riparian environment supports a distinctive "shrub thicket" vegetation.

- <u>Alpine tundra</u>: including rocky upland areas of sparse mat-forming or fell-field vegetation.

The species associated with each ecosystem at the Wainwright DEW Line installation have the potential to be exposed to COCs if exposure pathways are complete. If pathways are complete, the representative species selected are considered potential receptors. Figure 3-1, Section 3.2.3, Potential Exposure Pathways, presents a schematic model of the potential exposure pathways.

The Ecological Exposure Assessment segment of the risk assessment contains: the most common species found at the DEW Line installations in Section 3.2.1; the representative species and the rationale used for their selection in Section 3.2.2; a discussion of the exposure pathways

in Section 3.2.3; and a review of the habitat suitability for representative species in Section 3.2.4. Sections 3.2.5, 3.2.6, and 3.2.7 provide the methodology of the exposure assessment for representative plants, representative aquatic species, and representative birds and mammals, respectively. Life history tables, which provide species specific information for use in the exposure assessment, are included in Section 3.2.7.

3.2.1 Species of the Arctic Coastal Plain

The representative species in the ERA for the Wainwright installation were selected from those characteristic of the all the DEW Line installations along the Arctic Coastal Plain and are detailed in Sections 3.2.2.1 through 3.2.2.5.

The Wainwright installation is located along the northern boundary of the Arctic Coastal Plain. Hart Crowser (1987) and Woodward-Clyde (1993) have listed the species likely to occur along the coastal plain based on site-specific studies and a review of the literature. The marine zone, wet sedge meadows, tussock tundra, and riverine/riparian are the primary ecosystems found at the Wainwright installation. Alpine tundra is minimal at the site and is not evaluated further. Site-specific surveys of the ecosystems associated with the DEW Line installations have not been conducted for this risk assessment; however, a study investigating the abundance and distribution of Steller's and spectacled eiders was used (Alaska Biological Research 1994).

3.2.1.1 Plants. Plants commonly associated with the marine zone are sedges, grasses and forbs. *Carex subspathacea* and *C. aquatilis* are dominant plants in the coastal wetlands.

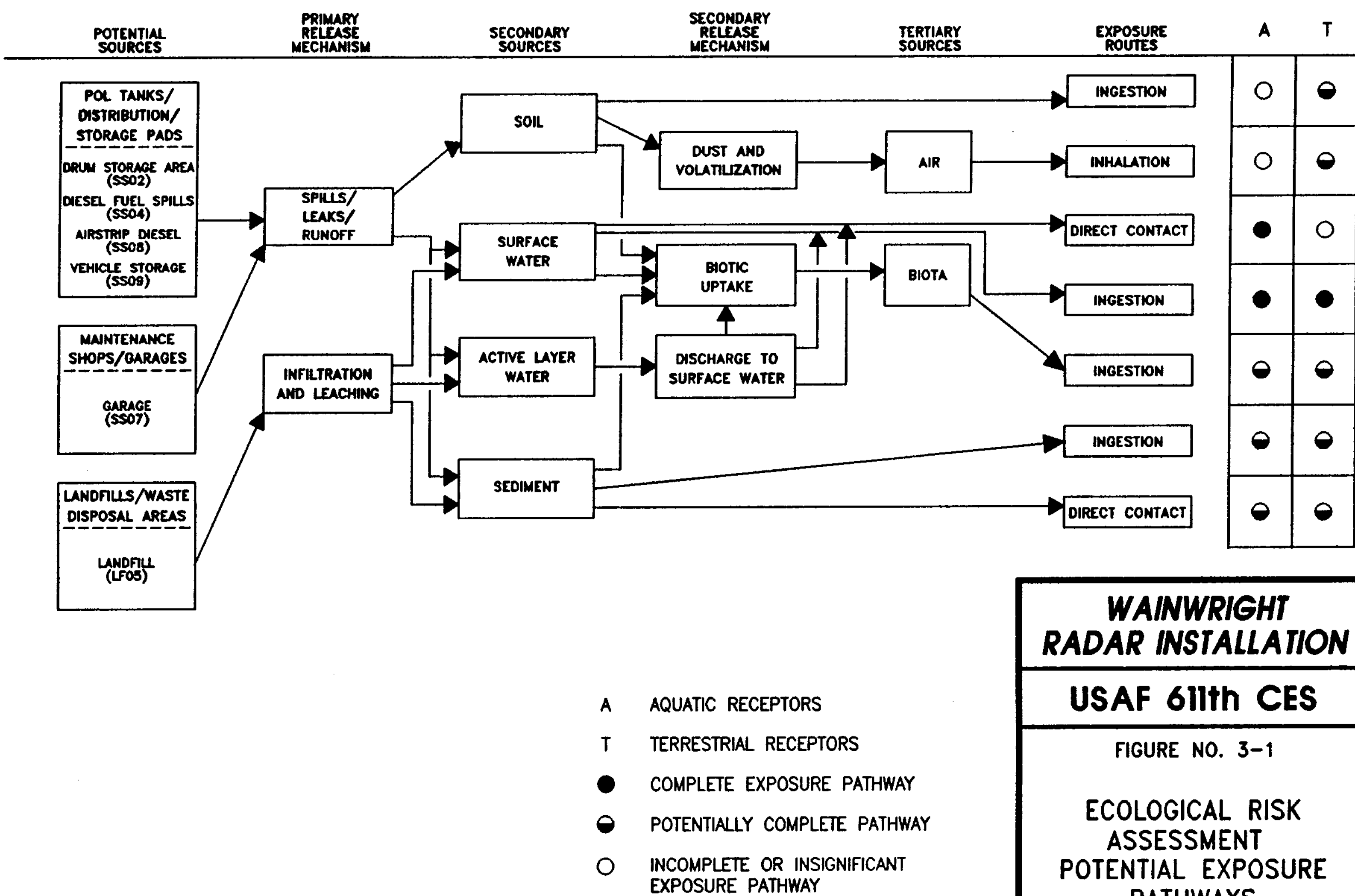
The wet sedge meadow (also known as "wet tundra") is characterized by a variety of sedges and grasses. Typical species include: cottongrass, *Eriophorum* spp.; tundra grass, *Dupontia fischeri*; and mosses, *Sphagnum* spp. Marsh marigold, *Caltha palustris*; and horsetail, *Equisetum spp.* may be found in wetter areas (Hart Crowser 1987).

The tussock tundra (or moist tundra) is drier than the wet sedge meadow/wet tundra association. Tussock-forming cottongrass is the dominant plant species. Grasses, sedges, dwarf shrubs, mosses, and lichens are scattered throughout the tussock complex. The species include: willows, *Salix* spp.; Labrador tea, *Ledum palustri*; blueberry and lingonberry, *Vaccinium* spp.; and lousewort, *Pedicularis* spp (NPRA Task Force 1978; Bergman et al. 1977).

Riverine/riparian systems are composed of a diversity of habitat types and species. The dominant plants here are shrubs with a scattered understory of grasses, herbs, and lower growing shrubs. Larkspur, *Delphinum brachycentrum*; cinquefoil, *Potentilla* spp.; bearberry, *Arctostaphylus* spp.; and wormword, *Artemesia arctica* are common species (NPRA Task Force 1978; Bergman et al. 1977).

3.2.1.2 Aquatic Organisms. Sixty-six species of fish inhabiting marine, estuarine, and freshwater systems have been identified in the arctic region (Hart Crowser 1987). Marine species inhabiting the nearshore and offshore waters include: boreal smelt, *Osmerus eperlanus*; Pacific herring, *Clupea harengus*; arctic cod, *Boreogagus saida*; and fourhorn sculpin, *Myoxocephalus*

DRAWING No. WAIN-FLO



WAINWRIGHT RADAR INSTALLATION

USAF 611th CES

FIGURE NO. 3-1

ECOLOGICAL RISK ASSESSMENT POTENTIAL EXPOSURE PATHWAYS

THIS PAGE INTENTIONALLY LEFT BLANK

quadricornis. Anadromous species using arctic rivers for spawning include the arctic cisco, *Coregonus autumnalis*; arctic char, *Salvelinus alpinus*; and occasional pink and chum salmon, *Onchorhynchus* spp. Lack of overwintering habitat is a significant limiting condition for both anadromous and freshwater fish of the arctic region. The principal freshwater fish found in the region are grayling, *Thymallus arcticus*; lake trout, *Salvelinus namaycush*; burbot, *Lota lota*; and nine-spine stickleback, *Pungitius pungitius* (Hart Crowser 1987).

Invertebrates that may be present in the waters and wet habitats of the Arctic Coastal Plain are well represented by the crustaceans (i.e., copepods, isopods, amphipods, and decapods).

3.2.1.3 Birds. There are approximately 180 species of birds seasonally associated with the habitats of the Arctic Coastal Plain. Of these, many are shorebirds and waterfowl using migratory corridors that pass through the Wainwright area (U.S. Air Force 1993b). Bird use of the coastal plain is highly seasonal and associated with typical avian breeding and migration cycles. Shoreline habitats are used significantly in association with molting, pre-migratory staging, and post breeding movement. These habitats are considered critical by the U.S. Fish and Wildlife Service (USFWS). Principal species include: glaucous gull, *Larus hyperboreus*; red phalarope, *Phalaropus fulicaria*; dunlin, *Calidris alpina*; loons, *Gavia* spp.; sandpipers, *Calidris* spp.; eiders, *Somateria* spp.; and geese, *Branta* spp. and *Chen* spp. Among the migratory passerine species using the coastal habitats are the Savannah sparrow, *Passerculus sandwichensis*; common and hoary redpolls, *Carduelis* spp.; snow bunting, *Plectrophenax nivalis*; and Lapland longspur, *Calcarius lapponicus* (Woodward-Clyde 1993).

3.2.1.4 Mammals. The mammalian fauna of the Arctic Coastal Plain and adjacent waters is relatively simple compared to fauna at lower latitudes. A review of species lists indicates a total of 38 species that commonly occur in the arctic; 11 of these are marine mammals (Hart Crowser 1987). A sampling of the terrestrial mammals geographically associated with the DEW Line stations, including Wainwright, consists of: brown lemming, *Lemmus trimucronatus*; masked shrew, *Sorex cinerus*; arctic fox, *Alopex lagopus*; red fox, *Vulpes vulpes*; weasels, *Mustela* spp.; tundra vole, *Microtus oeconomus*; caribou, *Rangifer tarandus*; and grizzly bear, *Ursus arctos* (Hart Crowser 1987; Woodward-Clyde 1993).

Marine mammals of the arctic coast include polar bear, *Ursus maritimus*; walrus, *Odobenus rosmarus;* six species of whales; and five species of seals. The most common of the whale and seal species are: beluga whale, *Delphinapterus leucas*; bowhead whale, *Balaena mysticetus*; gray whale, *Eschrichtius robustus*; ringed seal, *Phoca hispida*; and bearded seal, *Erignathus barbatus* (Hensel et al. 1984).

3.2.1.5 Threatened and Endangered Species. Species of the Arctic Coastal Plain and nearby waters that are protected by federal and state designations include: bowhead whale (endangered); fin whale, *Balaenoptera physalus* (endangered); Sei whale, *Balaenoptera borealis* (endangered); and hump-backed whale, *Megaptera novaengliae* (endangered). The gray whale has been delisted by the National Marine Fisheries Service as of 16 June 1994. Avian species include the spectacled eider, *Somateria fischeri* (threatened); and Steller's eider, *Polysticta stelleri* (candidate for threatened status). Based on the latest federal and state lists (June 1995) of

threatened and endangered plant species, no plant species at the coastal DEW Line installations are currently listed.

3.2.2 Representative Species

It is impractical to evaluate all of these receptors individually because of the great diversity of plants and animals at a given site. Thus, for ERAs, a set of "representative species" is selected for further evaluation. The representative species are selected based primarily on the species' likelihood of exposure based on their preferred habitat, feeding habits, and distribution of contaminants. Exposure pathways are shown in Figure 3-1 and discussed in Section 3.2.3. The abundance of a species, relative to the areal extent of the sites, is also considered. The representative species encompass a range of ecological niches in order to achieve the best characterization of the ecosystems being examined. In addition, species are selected, in part, as a result of the availability of toxicity, exposure, and life history information. Species that may be sensitive to environmental impacts, such as endangered or threatened species, are also evaluated.

For the DEW Line stations, groups of receptors are evaluated; including plants, aquatic invertebrates, fish, birds and mammals. Potential risks to representative species are estimated by evaluating sampling data for the relevant exposure media (i.e., soil, sediments and surface water). For plants, soil/sediment COC data are used to estimate potential uptake. For aquatic species, surface water COC concentrations are used to estimate exposure, and for the birds and mammals selected, exposures are estimated by evaluating their potential dietary intakes of COCs. No site-specific studies were conducted to determine exposure or toxicity levels at the installation.

The similarity of ecosystems at each of the installations allows the use of the same set of representative species for all installations. It may be possible that a representative species inhabits the general area of an installation, but does not occur specifically on the installation property. When and if this situation occurs, it will be noted. The receptors that are selected specifically as representative species for the Wainwright installation are listed in the paragraphs that discuss the representative groups (i.e., plants, aquatic organisms, birds, mammals, and threatened or endangered species). Table 3-3 presents the representative species for all the DEW Line installations, including endangered and threatened species that may be potentially exposed. The USFWS was consulted about the occurrence and selection of threatened and endangered species.

3.2.2.1 Representative Plants. Plants selected as representative species are: sedges, *Carex* spp.; willows, *Salix* spp.; and cottongrass, *Eriophorum* spp. These species are selected because they are abundant on all the sites, are important links in the trophic structure of the ecosystems of the arctic, and represent a major percentage of the primary production along the coastal plain. The blueberry, huckleberry, and lingonberry, *Vaccinium* spp., are evaluated because of their roles as forage plants and as subsistence species. All of these representative species are evaluated at the Wainwright installation.

TABLE 3-3. REPRESENTATIVE SPECIES AT THE DEW LINE INSTALLATION SITES

COMMON NAME	GENUS AND SPECIES
PLANTS	
Sedge	*Carex* spp.
Cottongrass	*Eriophorum* spp.
Willow	*Salix* spp.
Berries	*Vaccinium* spp.
AQUATIC ORGANISMS	
Water fleas	*Daphnia* spp.
Nine-spined stickleback	*Pungitius pungitius*
Arctic char	*Salvelinus alpinus*
BIRDS	
Lapland longspur	*Calcarius lapponicus*
Brant	*Branta bernicla*
Glaucous gull	*Larus hyperboreus*
Pectoral sandpiper	*Calidris melanotos*
MAMMALS	
Brown lemming	*Lemmus trimucronatus*
Arctic fox	*Alopex lagopus*
Barren-ground caribou	*Rangifer tarandus*
ENDANGERED AND THREATENED SPECIES	
Spectacled eider[a]	*Somateria fischeri*
Steller's eider[b]	*Polysticta stelleri*

a Threatened status.
b Candidate for threatened status, see text for explanation.

3.2.2.2 Representative Aquatic Invertebrates and Fish. The invertebrates selected as representative species are *Daphnia* spp (water fleas). The fish species chosen are the arctic char and the nine-spined stickleback. *Daphnia* spp. are abundant and represent a portion of the diet of the selected fish species (Johnson and Burns 1984; Wootton 1976), and toxicity information is readily available for them. The arctic char is a common anadromous species (exposed to both fresh and saltwater) and is a valuable recreation and subsistence resource (Johnson and Burns 1984). The nine-spined stickleback is a freshwater species that also uses brackish habitats, nests in aquatic vegetation, and is prey for other fish and bird species (Wootton 1976). Because the aquatic habitat at Wainwright is not suitable for arctic char, only the nine-spined stickleback and *Daphnia* will be evaluated in this ERA. No marine mammals are evaluated because there are no complete pathways for COCs (at concentrations that are of concern) to reach potential marine receptors.

3.2.2.3 Representative Birds. The representative avian species are Lapland longspur; brant, *Branta bernicla*; glaucous gull; and pectoral sandpiper, *Calidris melanotos*. The Lapland longspur is a passerine belonging to a terrestrial feeding guild (including sandpipers, turnstones, and phalaropes) (Custer and Pitelka 1978). The longspur's diet of insects and seeds (Custer and Pitelka 1978) makes it an important link in the arctic trophic web. The brant nests and molts among the numerous ponds in the tussock tundra and grazes on sedges and cottongrass (Palmer 1976). It is considered to be an important subsistence resource. The glaucous gull is a predatory scavenger that feeds on small mammals, young birds, carrion, and garbage, and breeds along the Arctic Coastal Plain (Farrand 1983). The pectoral sandpiper is an abundant shorebird that is primarily insectivorous and breeds on the Arctic Coastal Plain. The Lapland longspur, brant, glaucous gull, and pectoral sandpiper have potential exposure pathways at the Wainwright installation and will be evaluated in this ERA.

3.2.2.4 Representative Mammals. The representative species of mammals selected are the brown lemming, arctic fox, and the barren-ground caribou. The brown lemming is the predominant small mammal at all installations. The lemming consumes more vegetation than expected for an animal its size, due to its low assimilation efficiency, the low nutrient value of inter forage, and the high metabolic demands of the arctic environment (Chappell 1980). The arctic fox is selected as a representative species because it is ubiquitous along the coastal plain and its carnivorous diet (mostly lemmings) places it near the top of the trophic structure in the arctic. Eberhardt et al. (1982) note that in fall and winter, and to a lesser extent in summer, the arctic fox frequently uses areas near development; this tendency may expose the fox to contamination. Additionally, the fox, a relatively common furbearer, can be an important subsistence resource. The caribou is selected as a representative species because it uses areas on, or near, a number of the installations during migration, calving, and post-calving. In addition, the caribou is a significant subsistence resource for local people along the Arctic Coastal Plain (USFWS 1982; Cuccarese et al. 1984; and Hensel et al. 1984). The three mammal species discussed may be potentially exposed to COCs at the Wainwright installation and will be evaluated in this ERA.

3.2.2.5 Threatened and Endangered Species. The threatened and endangered species that potentially occur at the DEW Line installations are the spectacled eider and Steller's eider. The spectacled eider is federally listed as threatened, and Steller's eider is a candidate for listing as threatened. The U.S. Fish and Wildlife Service indicated that it was likely that Steller's eider

would be listed as threatened sometime in 1995 (Ambrose 1994 pers. comm.), but as of April 1995 a federal moratorium on additions to the threatened and endangered lists was in effect. When the ERA process for the DEW Line installations began, the arctic subspecies of peregrine falcon (*Falco peregrinus tundrius*) was listed as threatened, so it was included in the Barter Island ERA. In the interim, the peregrine's arctic subspecies has been delisted and is not considered in this or future ERAs, based on its current status. An Air Force contractor (Alaska Biological Research 1994) conducted surveys searching for spectacled and Steller's eiders on and near the DEW Line installations. The surveys concluded that neither species was present at the Wainwright installation. Currently, no threatened or endangered species are known to be in potential exposure pathways at the Wainwright installation and thus none are considered in this ERA.

3.2.3 Exposure Pathways

This section discusses potential exposure pathways for ecological receptors. In addition, methods used to quantify exposures to selected species of plants, aquatic organisms, birds, and mammals are presented. Quantitative estimates of exposure will be compared with toxicity reference values (TRVs) derived in Section 3.3 to estimate risks in the risk characterization section (Section 3.4).

Ecological receptors can be exposed to COCs through abiotic and biotic media. Potential exposure pathways for terrestrial and aquatic organisms are summarized in Figure 3-1. The following sections describe the potential exposure routes and a determination of pathways evaluated in the risk assessment.

Potential risks to representative species of plants from exposure to COCs in soil and water will be addressed. The most significant route of exposure for plants is direct contact with soil at the site, although a qualitative evaluation of the effects of COCs in surface water is presented in Section 3.4.1.

Aquatic organisms such as fish and invertebrates are primarily exposed through direct contact with surface water. Surface water is in direct contact with dermal surfaces as well as gills and other respiratory structures. Fish and invertebrates also may be exposed to COCs through ingestion of plant and animal items in the diet, direct contact with sediments, and incidental ingestion of sediments while foraging. Direct contact with surface water is the primary exposure route, however, and these secondary routes will not be evaluated for aquatic organisms.

Wildlife, such as birds and mammals, may be exposed to COCs through a variety of pathways including ingestion of surface water used for drinking, ingestion of plant and animal diet items, and incidental ingestion of surface soils and sediments while foraging. Wildlife species are not expected to be exposed to COCs via inhalation because the surface soils are well vegetated and moist during the growing season and frozen and/or snow covered the remainder of the year. Therefore this pathway is not evaluated in the ERA.

Insufficient toxicity and exposure information is available for the representative species to quantify exposures from dermal contact with soil or sediments; therefore, these pathways are not

quantitatively evaluated. Because soils and sediments represent potential pathways, total exposures for the representative species could be underestimated. This represents one of the uncertainties in this risk assessment discussed in Section 3.5.

3.2.4 Habitat Suitability for Representative Species

In order to assess the representative species' degree of exposure to the COCs, the habitat suitability of each of the six sites was evaluated. The habitat suitability evaluation considered the representative aquatic, avian, and mammalian species selected for evaluation at the Wainwright installation.

Human development and activities at the Wainwright installation affected the natural habitats available to the representative species. In some cases the activities probably deter wildlife use of the area and in other cases they may attract wildlife (e.g., arctic fox and gulls are attracted to a landfill). Because the Wainwright installation was deactivated in 1989, it is likely that the activity-related impacts are currently minimal and that ecological receptors, including the representative species, may use any or all of the sites at the Wainwright installation. As a result, all the sites at the Wainwright installation are evaluated in this ERA. In some cases, the media-specific samples have been taken at locations that do not represent suitable habitat for all the representative species (e.g., under-building sample locations obviously not suitable for caribou or surface water sample locations that are not accessible to fish species). This may result in an overestimate of exposure because sample data from all locations are used to calculate the average concentrations which, in turn, are used to estimate exposure for the representative species. These conditions will be noted in the risk characterization and uncertainty sections of the ERA (Sections 3.4 and 3.5, respectively).

The ERA is being conducted for the entire Wainwright installation, but only a portion of the facilities' estimated 482 hectares (ha) consists of potentially contaminated sites. The sites are estimated to total approximately 4.6 ha based on site maps. The spatial extent of the sites is considered when estimating the onsite dietary intake (IS) in Section 3.2.7.2. In general, based on professional judgement and onsite observation, but not on site-specific surveys, the installation provides habitat less suitable than nearby areas because of the numerous roads, gravel pads, and overall development.

3.2.5 Exposure Assessment for Representative Species of Plants

The harsh environment of the Arctic Coastal Plain imposes many restrictions on plant life. The presence of permafrost limits infiltration and percolation of water, so the water table is often at or above the surface. The vast majority of plant species are perennial, with much of their biomass (50 to 98 percent) underground (Raven et al. 1986). The potential pathways of contamination for plants are through the soil/sediment and surface water.

Carex spp., *Salix* spp., and *Eriophorum* spp. all store food reserves in rhizomes. Mychorrhizal fungi play an important role in the transport and delivery of nutrients to the rhizomes and the roots of these species. This underground system is most likely to be a response to the harsh above-ground arctic environment. As a result, surface water contaminated with chemicals that

are lighter than water (i.e., petroleum and its derivatives) does not present a hazard to the below-ground portion of plants. This has been shown experimentally by exposing arctic coast vegetation to petroleum products (Walker et al. 1978). The experiments showed that sedges, willows and cottongrass plants were not adversely affected by low to moderate amounts of petroleum in wet environments. Thus, soil/sediment will be considered the primary pathway of potential contamination for plants. The chemical concentration used in the Risk Characterization (Section 3.4) is the average concentration of the COC in the soil/sediments at the installation. A qualitative evaluation of the effect of potentially contaminated surface water on plants is presented in Section 3.4.1.

3.2.6 Exposure Assessment for Representative Aquatic Organisms

Organisms that dwell in an aquatic environment are exposed to chemicals contained in the water column. For this reason, the exposure assessment considers the concentrations in surface water to be the exposure concentrations to aquatic organisms. As described in Section 3.2.3, the primary exposure route for aquatic organisms is direct contact with surface water, and as a result, *Daphnia* spp. are not evaluated for contact with or ingestion of sediments. The risk assessment compares the average concentration of the COCs found in surface waters to toxicity data for the representative aquatic species to determine the risk.

3.2.7 Exposure Estimates for Representative Bird and Mammal Species

Exposure estimates for the representative species of mammals and birds (expressed as a unit of chemical ingested per unit of body weight) are based on their total exposure to COCs from diet, soils, and surface water using the following equation:

$$EE = [(FI \times CF) + (WI \times CW) + (SI \times CS \times ROA)] \times UCF \times IS / BW$$

where:

EE	=	estimated exposure (mg/kg-bw/day).
FI	=	food intake rate (g/day); rates are derived in the life history tables (3-5 through 3-13). Diets (both vegetable and animal components) are proportioned according to the diet composition information in the life history tables and are presented below.
CF	=	chemical concentration in food (mg/kg); based on concentrations for each group of food items.
WI	=	water intake rate (L/day); rates are derived in the life history tables.
CW	=	chemical concentration in water (μg/L); see Section 3.1 for calculations of concentrations.
SI	=	soil/sediment intake rate (g/day); based on a percentage of food intake.
CS	=	chemical concentration in soil/sediment (mg/kg); see Section 3.1 for calculations of concentrations.
ROA	=	relative oral availability; default to 1.0 (lack of information). This value assumes that the bioavailability of the chemical in the test medium is the same as for the medium onsite.

UCF	=	0.001; unit conversion factor used to convert g/day to mg/day (FI and SI) and L/day (WI) to ml/day, to ensure EE is reported in mg/kg-bw/day.
IS	=	fraction of dietary intake at potentially contaminated sites (by weight).
BW	=	body weight (kg).

In the case of species that have partial herbivorous dietary intakes, the CF x FI phrase in the equation is multiplied by the proportion of vegetation in their diet (these calculations are presented in Appendix C, Concentration in Food Calculations). Those species and their respective proportions are: Lapland longspur, 0.25; brant, 0.90; glaucous gull, 0.10; and pectoral sandpiper, 0.10 (see the life history tables for references regarding the proportion of vegetation in the species' diets). The estimated exposure calculations for bird and mammal receptors are presented in Appendix D.

3.2.7.1 Potential Bioaccumulation of COCs in Representative Species. The potential risks from ingestion of COCs in dietary items are difficult to determine because of the complexity of the trophic web. Inputs to the exposure estimate equation include concentrations of contaminants in water and soil, ingestion rates for water, food, and soil, the relative use of the potentially contaminated sites compared to the representative species' normal range, and body weight. In the case of higher level consumers, the food ingested may be from different levels of the trophic web. For example, a contaminant may be taken up by a plant, which is consumed by a lemming, which is then eaten by an arctic fox. The amount of contaminant to which the fox is exposed is not readily quantified without supporting empirical data at each trophic level. For this reason, and because of the lack of data to quantify bioaccumulation, the risk assessment does not account for bioaccumulation in the animal portion of the trophic web. This uncertainty is tempered by the "hot spot" nature of the distribution of the COCs. It is possible that representative species may be exposed to these "hot spots" occasionally, but unlikely that their entire exposure will occur at these spots. Use of average concentrations overestimates the potential exposure of representative species (this is discussed in more detail in the ERA Uncertainty Analysis, Section 3.5.1). Furthermore, the likelihood of predators repeatedly taking prey that were exposed to a COC "hot spot" is low. For example, the arctic fox ranges over such a wide area, that any COCs to which the fox would be exposed via bioaccumulation would represent only a very small proportion of its overall exposure.

Further, most of the COCs at the Wainwright installation are VOCs that are unlikely to bioaccumulate. For illustrative purposes, bioconcentration factors (BCFs) were calculated (Veith et al. 1979) for the organic COCs and are presented in Appendix E and summarized in Table 3-4. Because they are low, the exposure estimates for organic chemicals do not include potential bioaccumulation of COCs in the animal portion of the trophic web. It is unlikely that the organic chemicals will bioaccumulate (based on the concentrations reported in the soil and water) such that the exposure estimates would exceed, or even approach, the TRVs.

The metals of concern at Wainwright (i.e., aluminum, cadmium, iron, lead, manganese, vanadium, and zinc) are evaluated for plant uptake quantitatively, as discussed in Section 3.2.5. It is not possible to quantify bioaccumulation of metals in the animal portion of the trophic web without sample concentrations at each level of consumer. This problem is addressed qualitatively in the following discussions of the bioaccumulative properties of the metals of concern.

Aluminum is not likely to bioaccumulate because, taken orally, it is poorly absorbed through the gastrointestinal tract. Most ingested aluminum leaves an animal's body in the feces, and the little that does enter the bloodstream is excreted in the urine (ATSDR 1990a).

Cadmium has demonstrated bioaccumulative properties in several experimental studies (Eisler 1985). However, cadmium was detected at a single location at the Wainwright installation: sample SD02 at the Landfill. Sample SD02 is downgradient from the Landfill and also downgradient from sample SD01. Sample SD01 was not analyzed for metals, so it is difficult to speculate whether concentrations at sample SD02 are anomalous. There may be a limited risk associated with this location; however, because contamination is likely to be limited to one or more "hot spots", the associated ecological risk is low.

Iron Information about the bioaccumulation of iron is not available in the literature, but it is likely that the metabolic processes that make use of iron will prevent undue bioaccumulation because iron is an essential nutrient.

Lead tends to accumulate in bone (Talmadge and Walton 1991), so ingestion of animal tissue would not contribute greatly to increased lead concentrations. Food chain biomagnification of lead is uncommon in terrestrial communities (Eisler 1988). Kraus (1989) showed that in environments that were high in lead, the concentration of lead in insects and the tissues of insectivorous birds was low. Thus, lead is not likely to bioaccumulate to a degree that could contribute to risk at Wainwright.

Manganese and **zinc** are considered essential nutrients (ATSDR 1990b, 1989; Eisler 1993). It is not likely that the concentrations of these metals found at the Wainwright installation could bioaccumulate because animal systems are conditioned to regulate these minerals for metabolic use.

TABLE 3-4. BIOCONCENTRATION FACTORS FOR SELECTED ORGANIC COMPOUNDS IN WATER

CHEMICAL	Log K_{ow}	BCF
DRPH	5.30	6,238
Ethylbenzene	3.15	146
Naphthalene	3.36	211
Tetrachloroethene	2.53	49
Trimethylbenzene	3.78	439
Xylene	3.16	149

Note: BCF calculated from Log K_{ow} according to the following equation:
Log BCF = 0.76 Log K_{ow} - 0.23 (Veith et al. 1979 in Spacie and Hamelink 1985).
K_{ow} = octanol/water partition coefficient.

Vanadium was detected only once in surface water at the Vehicle Storage Area (sample SW01). Studies focusing on vanadium bioaccumulation in aquatic ecosystems have shown very little evidence of amplification of vanadium in different trophic levels (Lee 1983 in Nriagu 1983). It is unlikely to present a bioaccumulative risk.

3.2.7.2 Estimation of Percent Ingested Onsite. The size of the areas that the representative species use, and hence their potential exposure to COCs, varies greatly. Generally, a species' home range is used to characterize the size of the area it uses on a regular basis (disregarding migration and dispersal). This information, combined with the extent of the potentially contaminated sites, can be used to estimate the percent of dietary intake from the sites.

This estimate is referred to as the "percent of dietary intake at sites" (IS) value in the exposure estimate equation. The IS value is represented by the ratio of the total area of the sites (4.6 ha) to the reported home range size (or converted population density values) for the representative species. When home range information for a species was not available, population density values were converted to estimate the area used by the species. The representative species are most likely at Wainwright during, or directly after, the breeding season, when many species become territorial. These territories represent the area used by the species and can be estimated from population density. This presents an added degree of uncertainty (see Section 3.5.3).

If the home range (or converted population density value) is less than the total areal extent of the sites (4.6 ha), the maximum value for IS is 1.0 because it is possible that a species could meet all its dietary intake needs within the potentially contaminated areas. The IS values for the representative bird and mammal species are given below. Note that this is a conservative estimate because the 4.6 ha site size assumes that is the only area used. Obviously, the species would use the suitable areas between the potentially contaminated sites and correspondingly reduce potential exposure.

Birds. Lapland longspur. IS = 0.5; Derksen et al. (1981) report a breeding density of 38.6 birds/km^2. This corresponds to about 1 bird/2.6 ha. Potentially, the longspur could meet all its dietary demands within the potentially contaminated sites. Nevertheless, an IS value of 0.5 is used because the longspur prefers drier upland habitat over the wetter areas where the majority of the contaminant pathways occur.

Brant. IS = 0.25; density of breeding pairs reported by Derksen et al. (1981) is 5.0 birds/km^2. At this density of 1 brant/20 ha, the total extent of the potentially contaminated sites is about 25 percent of the area a brant might use.

Glaucous gull. IS = 0.04; the density for the glaucous gull is reported by Derksen et al. (1981) as 0.8 birds/km^2. This density, about 1 gull/125 ha, yields an IS value of about 0.04 when compared to the total extent of the potentially contaminated sites.

Pectoral sandpiper. IS = 1.0; the density of the pectoral sandpiper along the Arctic Coastal Plain is reported by Derksen et al. (1981) as 22.4 birds/km^2. This density equates to one sandpiper/4.5 ha, and a corresponding IS value of approximately 1.0.

Mammals. Brown lemming. IS = 0.5; the lemming's home range is reported as 0.5 ha (Nowak 1991). It is possible that several lemmings may consume all their dietary needs within the bounds of a site. However, the lemming is not likely to use the wetter sites (which constitute well over 50 percent of the total extent of the sites), where the majority of the contaminant pathways are located. The sites are mostly gravel pads that have been constructed for development purposes, support little or no vegetation, and offer a poor matrix for the lemming to use for burrowing. For these reasons the IS used for the brown lemming is 0.5 rather than 1.0.

Arctic fox. IS = 0.01; the home range of the fox is extremely variable. Eberhardt et al. (1982) report a home range of 3.7 to 20.8 km^2 for juvenile and adult arctic foxes, respectively. Even the lower end of this range (3.7 km^2 or 370 ha) is approximately 100 times greater than the extent of the sites, hence the IS value of 0.01.

Caribou. IS = 0.01; caribou are highly mobile, covering large distances during their movements to and from calving grounds and in their constant search for suitable forage. They may range over thousands of km/year, and as a result their is no accurate estimate of their home range. Based on knowledge of the caribou's habits and professional judgement, a very conservative estimate of the likelihood of caribou using the potentially contaminated sites is 1 percent, or a corresponding IS value of 0.01.

3.2.7.3 Exposure Assessment for Representative Species of Birds. In this section the methods for quantifying exposures to the selected representative species of birds are presented.

In order to estimate exposures of the representative species of birds, life history information was compiled for the selected species. This information includes: occurrence at the DEW Line sites, habitat, average body weight, estimated food intake rate, estimated water intake rate, diet composition, and home range and/or population density.

Plant uptake of contaminants has been quantified for use in the exposure estimations for herbivores (bird and mammal species). Herbivores are potentially exposed to contamination directly from ingestion of soil and water intake as well as through their diet. The dietary plant component (CF in the exposure estimate equation) is calculated by multiplying the contaminant's soil concentration by the BCF, B_v. B_v is defined as the ratio of the concentration in above-ground parts of a plant (mg of compound/kg of dry plant) to the concentration in soil (mg of compound/kg of dry soil). The B_v can be used to predict the level of a potential contaminant taken up by a plant, and this information can then be used to assess the potential transport of the contaminant in the trophic web.

The uptake of metals by plants is quantified using the B_v values in Baes et al. (1984). The approach for organic chemicals is basically the same, except that the B_vs for organic chemicals are derived using a regression equation (Travis and Arms 1988). The equation is:

$$\log B_v = 1.588 - 0.578(\log K_{ow})$$

where:

B_v = the BCF (unitless) and

K_{ow} = the octanol-water partition coefficient of the chemical (mol/m^3 / mol/m^3).

In order to calculate the potential uptake of DRPH by plants, the K_{ow} of diesel fuel was estimated using equation 2-3 in Lyman et al. (1982):

$$\log S = -0.922 \log K_{ow} + 4.184$$

where:

S = solubility (mg/L) and

K_{ow} = octanol/water partition coefficient (mol/m^3 / mol/m^3).

This equation estimates the solubility of an organic chemical in water. However, it may also be manipulated arithmetically to calculate the log K_{ow} based on the known solubility:

$$\log K_{ow} = \frac{\log S - 4.184}{-0.922}$$

The solubility of diesel fuel (0.2 mg/L) (Custance et al. 1992) was used to calculate a log K_{ow} for diesel fuel of 5.3.

Life history information for the Lapland longspur, brant, glaucous gull, and pectoral sandpiper is presented in Tables 3-5 through 3-8.

Information is not available on the daily food intake rate (grams/day) and water intake rate (liters/day) for the representative bird species in the arctic habitat. Therefore this information was estimated using regression equations associated with body weight (Calder and Braun 1983). The severity of the arctic climate may impose higher metabolic demands on animals. As a result, the food and water intake rates should be considered to be estimates only and their uncertainty should be kept in mind. The food intake rate was estimated using Nagy's (1987) equations:

Passerine birds (i.e., Lapland longspur):

$$FI \text{ (g/day dry matter)} = 0.141 \times (\text{body weight in kilograms})^{0.850}$$

All other birds:

$$FI \text{ (g/day dry matter)} = 0.0582 \times (\text{body weight in kilograms})^{0.651}$$

The water intake rate was estimated using the regression equation developed by Calder and Braun (1983):

All birds:

$$WI \text{ (liters/day)} = 0.059 \times (\text{body weight in kilograms})^{0.67}$$

As animals forage they may incidentally ingest soil and sediment particles. The average concentration of contaminants in soil/sediment can be multiplied by the amount of soil/sediment ingested to estimate the potential intake of contaminants by this route. Soil intake rates have been reported for just a few wildlife species (Beyer et al. 1994). The soil ingestion rates for the representative species are extrapolated from Beyer et al. (1994) from similar species with reported values. The percentages reported are of the total weight of dietary intake. Table 3-9 lists the representative bird species, the species used as surrogates, and the estimated percentages of

TABLE 3-5. LIFE HISTORY INFORMATION FOR THE LAPLAND LONGSPUR, *Calcarius lapponicus*

PARAMETER	VALUE	NOTES	REFERENCE
OCCURRENCE AT DEW LINE SITES	seasonal breeder at all arctic coastal radar installations	dominant breeding passerine	U.S. Air Force 1993b
HABITAT	breeds on arctic coastal tundra		Scott 1983
BODY WEIGHT	27.3 g (0.027 kg)	mean of 68 specimens	Dunning 1984
FOOD INTAKE RATE	6.5 g/day dry matter	$FI=0.141(BWkg)^{0.850}$	Nagy 1987
WATER INTAKE RATE	0.005 liters/day	$WI=0.059(BWkg)^{0.67}$	Calder and Braun 1983
DIET COMPOSITION	during breeding (June and July): insects (craneflies); pre- and post-breeding (May and August): seeds (grasses); average 25 percent vegetation in diet	passerine member of insectivorous foraging guild which includes shorebirds	Custer and Pitelka 1978
POPULATION DENSITY	38.6/km^2	varies with changing predation pressures	Derksen et al. 1981

TABLE 3-6. LIFE HISTORY INFORMATION FOR THE BRANT, *Branta bernicla*

PARAMETER	VALUE	NOTES	REFERENCE
OCCURRENCE AT DEW LINE SITES	seasonal, breeds at or near all arctic coastal radar installations	breeding, migratory sp., subsistence sp.	U.S. Air Force 1993b
HABITAT	breeds on Arctic Coastal Plain	prefers low, barren, wet, coastal terrain	Palmer 1976
BODY WEIGHT	1,305 g (1.305 kg)	mean of 791 specimens	Dunning 1984
FOOD INTAKE RATE	69.2 g/day dry matter	$FI=0.0582(BWkg)^{0.651}$	Nagy 1987
WATER INTAKE RATE	0.07 liters/day	$WI=0.059(BWkg)^{0.67}$	Calder and Braun 1983
DIET COMPOSITION	sedges, grasses; average 90 percent vegetation in diet	some insects during breeding (June and July)	Palmer 1976
POPULATION DENSITY	5.0/km^2	average from three coastal sites	Derksen et al. 1981

TABLE 3-7. LIFE HISTORY INFORMATION FOR THE GLAUCOUS GULL, *Larus hyperboreus*

PARAMETER	VALUE	NOTES	REFERENCE
OCCURRENCE AT DEW LINE SITES	seasonal breeder and migrant at all arctic coastal radar installations	relatively common along arctic coast	Woodward-Clyde 1993
HABITAT	coastal tundra, lakes, ponds, and marine environment	breeds on arctic coast	Farrand 1983
BODY WEIGHT	1,445 g (1.445 kg)	mean of 65 specimens	Dunning 1984
FOOD INTAKE RATE	74 g/day dry matter	$FI=0.0582(BWkg)^{0.651}$	Nagy 1987
WATER INTAKE RATE	0.08 liters/day	$WI=0.059(BWkg)^{0.67}$	Calder and Braun 1983
DIET COMPOSITION	small fish, birds, insects, crustaceans, mollusks, garbage; average 10 percent of vegetation in diet	predatory scavenger	Martin et al. 1961
POPULATION DENSITY	$0.8/km^2$	average from three coastal sites	Derksen et al. 1981

TABLE 3-8. LIFE HISTORY INFORMATION FOR THE PECTORAL SANDPIPER, *Calidris melanotos*

PARAMETER	VALUE	NOTES	REFERENCE
OCCURRENCE AT DEW LINE SITES	seasonal breeder at all arctic coastal radar installations	abundant on Arctic Coastal Plain	Woodward-Clyde 1993
HABITAT	grassy margins of wet meadows, marshes, riparian areas, ponds	nests hidden on well-drained grassy sites	Scott 1983; Martin et al. 1961
BODY WEIGHT	79 g (0.079 kg)	mean of 35 specimens	Dunning 1984
FOOD INTAKE RATE	11.2 g/day dry matter	$FI=0.0582(BWkg)^{0.651}$	Nagy 1987
WATER INTAKE RATE	0.01 liter/day	$WI=0.059(BWkg)^{0.67}$	Calder and Braun 1983
DIET COMPOSITION	insects, mollusks, crustaceans, worms, vegetable debris; average 10 percent vegetation in diet	craneflies are major diet component	Martin et al. 1961; Pitelka 1959
POPULATION DENSITY	$22.4/km^2$	average from three coastal sites	Derksen et al. 1981

soil ingested. Species that forage directly in the soil or sediment, such as the sandpiper or goose, show relatively high percentages of soil in their diet. The Lapland longspur does not have appropriate surrogate species with soil ingestion data. Although the longspur is in the same foraging guild as sandpipers (which incidentally ingest relatively large amounts of soil), the longspur takes insects from the soil surface or gleans its prey from vegetation (Custer and Pitelka 1978), thus minimizing its soil intake, which is reflected in the estimate of soil ingestion (less than two percent of diet by weight). The glaucous gull ingests stones and sand as a mechanical addition (to aid in digestion) to its diet (Belopol'skii 1961) and this contributes to its soil/sediment intake. For those species without a suitable surrogate (i.e., Lapland longspur) or whose soil ingestion rate is reported as less than two percent, a value of two percent of dietary intake (by weight) was used to calculate the exposure estimates.

3.2.7.4 Exposure Assessment for Representative Species of Mammals. This section assesses exposure to contaminants for the selected representative species of mammals. Tables 3-10 (brown lemming), 3-11 (arctic fox), and 3-12 (caribou) present life history data that are used to calculate exposure estimates for the representative species. Home range and/or population density has been listed for the representative mammal species, depending on appropriateness and availability.

Information on daily food intake rates for the arctic fox and caribou is not available. The rates have been estimated using regression equations associated with average body weights and metabolic rates (Nagy 1987). The food intake rates for the fox and caribou were estimated using the following equations, developed for placental mammals in general and for herbivorous mammals, respectively (Nagy 1987).

arctic fox: using equation for placental mammals in general

$$\text{FI (g/day dry matter)} = 0.0687 \times (\text{body weight in kilograms})^{0.822}$$

caribou: using equation for mammalian herbivores

$$\text{FI (g/day dry matter)} = 0.0875 \times (\text{body weight in kilograms})^{0.727}$$

Because of very low assimilation efficiencies, the low nutrient content of winter forage, and the high metabolic demands in arctic habitats (Chappell 1980), the equation for food intake rate significantly underestimates the rate for the brown lemming. A more appropriate rate for the brown lemming of 45 g/day is reported by Chappell (1980) (using the highest value in the reported range of 24 - 45g/day).

The rates for water intake of the representative mammals were estimated using the regression equation generated by Calder and Braun (1983) because of the unavailability of species-specific information in the literature. The equation is:

$$\text{WI (liters/day)} = 0.099 \times (\text{body weight in kilograms})^{0.90}$$

TABLE 3-9. SOIL INGESTION ESTIMATES FOR REPRESENTATIVE BIRD SPECIES

REPRESENTATIVE SPECIES	SURROGATE SPECIES	ESTIMATED PERCENT OF SOIL IN DIET	ESTIMATED g/day SOIL IN DIET
Lapland longspur	no suitable surrogate	<2.0	0.1
Brant	Canada goose	8.2	5.7
Glaucous gull[a]	Siberian glaucous gull	7.6	5.6
Pectoral sandpiper	four sandpiper species (average)	18.1	2.0

a Information from Belopol'skii 1961.
Source: Beyer et al. 1994

TABLE 3-10. LIFE HISTORY INFORMATION FOR THE BROWN LEMMING, *Lemmus trimucronatus*

PARAMETER	VALUE	NOTES	REFERENCE
OCCURRENCE AT DEW LINE SITES	resident at all arctic coastal radar installations	dominant small mammal	U.S. Air Force 1993b
HABITAT	tundra and alpine meadows	nests aboveground in winter, below in summer	Burt and Grossenheider 1976
BODY WEIGHT	55 g (0.055 kg)		Chappell 1980
FOOD INTAKE RATE	24 - 45 g/day dry matter	has low assimilation efficiencies (31 - 36 percent), variation also related to seasons	Chappell 1980
WATER INTAKE RATE	0.007 liters/day	$W_0I=0.099(BWkg)^{0.9}$	Calder and Braun 1983
DIET COMPOSITION	sedges, grasses, lichens, roots, leaves, bark, berries		Nowak 1991
HOME RANGE SIZE (AVG)	0.5 ha (females) 1.0 ha (males)	0.5 ha used in assessment	Nowak 1991
POPULATION DENSITY	0 to 325/ha	populations have large fluctuations on a three to five year cycle; currently populations are low	Nowak 1991; Snyder-Conn 1994

TABLE 3-11. LIFE HISTORY INFORMATION FOR THE ARCTIC FOX, *Alopex lagopus*

PARAMETER	VALUE	NOTES	REFERENCE
OCCURRENCE AT DEW LINE SITES	resident at all arctic coastal radar installations	ubiquitous	U.S. Air Force 1993b
HABITAT	tundra and coastal plain	dens in sandy mounds >1 m high	Chesemore 1967
BODY WEIGHT	4950 g (4.95 kg)		Burt and Grossenheider 1976
FOOD INTAKE RATE	256 g/day dry matter	$FI = 0.0687(BWkg)^{0.822}$	Nagy 1987
WATER INTAKE RATE	0.42 liters/day	$WI = 0.099(BWkg)^{0.90}$	Calder and Braun 1983
DIET COMPOSITION	brown lemming (summer), nesting birds, carrion, seal pups, non-food items	brown lemming in >85 percent of all scats, n=224	Chesemore 1967; Nowak 1991
HOME RANGE SIZE (AVG)	20.8 km^2 adult 3.7 km^2 juvenile (<1 yr)	adult range used in assessment	Eberhardt et al. 1982

TABLE 3-12. LIFE HISTORY INFORMATION FOR THE BARREN-GROUND CARIBOU, *Rangifer tarandus*

PARAMETER	VALUE	NOTES	REFERENCE
OCCURRENCE AT DEW LINE SITES	seasonal, at or near all arctic coastal radar installations during migrations	some sites used for calving	U.S. Air Force 1993b
HABITAT	tundra in summer, open coniferous forest in winter	varies much, related to migration	Burt and Grossenheider 1976
BODY WEIGHT	95,500 g (95.5 kg)	mean for adults, male and female	Nowak 1991
FOOD INTAKE RATE	2400 g/day (2.4 kg) dry matter	$FI=0.0875(BWkg)^{0.727}$	Nagy 1987
WATER INTAKE RATE	6.0 liters/day	$WI=0.099(BWkg)^{0.90}$	Calder and Braun 1983
DIET COMPOSITION	willows, sedges, cottongrass, lichens	selection based on plant phenology	Skogland 1980; White and Trudell 1980
POPULATION DENSITY	1.41 km^2 0.31 km^2 4.53 km^2	undisturbed calving area within 1 km of road within 5 - 6 km of road	Cameron et al. 1992

Incidental soil intake was evaluated for mammals in the same manner as for birds (Section 3.2.7.3). Table 3-13 shows the percent of soil ingested for the representative mammal species.

3.3 ECOLOGICAL TOXICITY ASSESSMENT

This section presents toxicity information for each COC in surface water and soils/sediments. The COCs in surface water (Section 3.1.1) are limited to aluminum, iron, manganese, and zinc.

The COCs in soils/sediments (Section 3.1.2) include both organic and inorganic chemicals. The COCs in soils/sediments are DRPH, GRPH, RRPH, ethylbenzene, xylenes, tetrachloroethene, naphthalene, trimethylbenzene, aluminum, iron, lead, manganese, and zinc. Sections 3.3.1 through 3.3.13 discuss the toxicity of all COCs to the receptor groups. Section 3.3.14 presents the methodology for derivation of TRVs used for this ERA.

3.3.1 Petroleum Hydrocarbons

Section 3.1 presents the COCs for sites at the Wainwright installation. DRPH, GRPH, and RRPH were identified as COCs in soils/sediments. This section is a discussion of the chemical differences between DRPH, GRPH, and RRPH and the toxicity of these three petroleum mixtures.

Crude petroleum contains thousands of different chemical compounds. Gasoline and diesel fuel are refined petroleum products. The composition of gasoline and diesel fuel depends not only on the origin of the crude oil from which the gasoline is derived, but also the process technique and the blending scheme (Von Burg 1993). Once gasoline or diesel fuel is released to the environment, weathering and volatilization further alter its composition.

Gasoline is a complex, highly variable mixture of petroleum hydrocarbons containing 3 to 21 carbon atoms; however, compounds with 4 to 12 carbon atoms predominate. Gasoline is detected with the petroleum hydrocarbon analysis as GRPH. The following chemical classes are detected as GRPH: paraffins (straight-chained alkanes), olefins (straight-chained alkenes),

TABLE 3-13. SOIL INGESTION FOR REPRESENTATIVE MAMMAL SPECIES

REPRESENTATIVE SPECIES	SURROGATE SPECIES	ESTIMATED PERCENT OF SOIL IN DIET	ESTIMATED g/day SOIL IN DIET
brown lemming	prairie dog (average of two species)	6.2	1.2
arctic fox	red fox	2.8	7.2
caribou	elk	<2.0	48

Source: Beyer et al. 1994

naphthenes (cycloalkanes and alkenes), and aromatic hydrocarbons (alkylbenzenes and polynuclear) (VonBurg 1993). Although GRPH are generally in the range of 4 to 12 carbon atoms, the laboratory that conducted the analyses for Wainwright detected GRPH with 6 to 9 carbon atoms. As many as 140 compounds have been identified as constituents of gasoline; however, most of the toxicity is attributable to a few compounds of which benzene is the most toxic. Diesel fuel is also a complex, variable mixture of the same classes of compounds containing 6 to 21 carbon atoms. Diesel fuel is detected with a petroleum hydrocarbon analysis as DRPH. The laboratory that analyzed samples for Wainwright detected DRPH with 10 to 24 carbons atoms. As many as 45 compounds have been identified as constituents of diesel fuel (Von Burg 1993). RRPH could include many different types of chemicals, although the majority of molecules would include 24 carbon atoms or more.

Table 3-14 presents the chemical classes and weight percent for GRPH and DRPH. Generally, gasoline contains more aromatic compounds and simple chained alkanes, whereas diesel fuel is characterized by cycloparaffins (or cycloalkanes). Both gasoline and diesel fuel will be affected by the environment. Weathering will change the chemical composition of petroleum, and concentrations of aromatic compounds such as benzene will decrease as a result of volatilization.

TABLE 3-14. CHEMICAL CLASSES OF GRPH AND DRPH

CHEMICAL CLASS	WEIGHT PERCENT (%)[a]
GRPH[b]	
Normal paraffins (n-alkanes)	19.3-38.4 (28.8)
Isoparaffins (isoalkanes)	11.5-50.3 (30.9)
Naphthenes (cycloparaffins or cycloalkanes)	1.0-2.8 (1.9)
Aromatics (e.g., benzene, toluene, pyrene)	9.7-54.7 (32.2)
DRPH[c]	
Normal paraffins (n-alkanes)	5.6
Isoparaffins (isoalkanes)	11.1
Naphthenes (cycloparaffins or cycloalkanes)	46.3
Aromatics (e.g., benzene, toluene, pyrene)	33.3
Nitrogen, sulfur and oxygen compounds	3.7

a Average shown in parentheses.
b Heath et al. 1993.
c Weeks et al. 1988.

Available toxicity test data have been derived from pure, fresh product, and therefore the applicability to the weathered product encountered at Wainwright is uncertain. Gasoline is the most studied of the petroleum products; however, most data are based on inhalation studies. Gasoline was classified by EPA (1992c) as a Group C (possible human) carcinogen, whereas diesel oil was classified as Group D (not classifiable as to human carcinogenicity). Presumably, this classification of gasoline is due to benzene which, under the conditions of environmental exposure, would volatilize more rapidly than any other constituent. The gasoline and diesel petroleum hydrocarbon data from surface water and sediment samples collected at Wainwright indicate that benzene was not detected frequently at concentrations above either background or action levels. Physical-chemical data from the literature indicates that TPH in soil would reflect all constituents with eventual loss of aromatic (e.g., BTEX) components first, lighter alkanes second, lighter polynuclear aromatic hydrocarbons (PAHs) third, followed by naphthalenes. For an old diesel or petroleum spill, TPH measurements may reflect predominantly trace amounts of high molecular-weight PAHs or higher molecular-weight branched alkanes [Massachusetts Department of Environmental Protection (MDEP) 1993].

For the purposes of ranking the toxicity of GRPH, DRPH, and RRPH, it was assumed that BTEX and lighter-weight alkanes are significantly weathered from exposure to the arctic environment, and that toxicity is more dependent upon noncarcinogenic endpoints associated with alkanes, alkenes, and cycloalkanes. In addition, the toxicity of DRPH and RRPH is associated with the PAH content. However, at Wainwright, the only PAH detected was naphthalene, a chemical considered to be noncarcinogenic. Carcinogenic PAHs such as benzo(a)pyrene were not detected. MDEP (1983) reviewed the noncarcinogenic toxicological endpoints in laboratory animals for diesel fuel and gasoline and determined that diesel fuel was an order of magnitude more toxic than gasoline. Although other sources indicate that the toxicity of alkanes and cycloalkanes is similar (Armstrong Laboratory 1994; Sax and Lewis 1989). A review of the Wainwright data indicates that DRPH are present at higher concentrations than either GRPH or RRPH. Specifically, average concentrations of DRPH were approximately 228 times higher than average concentrations of GRPH and 1.5 times higher than RRPH. As a result, based on the MDEP review and the chemical data reported for the Wainwright surface water and soil/sediment samples, DRPH were used as a conservative representation of ecological risks from petroleum hydrocarbon contamination (i.e., GRPH and RRPH).

As discussed above, diesel fuel is comprised of a complex mixture of paraffins (straight-chained alkanes), olefins (straight-chained alkenes), naphthenes (cycloalkanes and alkenes), and aromatic (alkylbenzenes, and polynuclear) petroleum hydrocarbons containing 6 to 21 carbon atoms. Hydrocarbons containing 8 to 18 carbon atoms predominate (Von Burg 1993). There are six grades of diesel fuel (Diesel Oil No. 1, Diesel Oil No. 2, Diesel Oil No. 4, Fuel Oil No. 1, Fuel Oil No. 2, and Home Heating Oil) (Von Burg 1993). The specific components of diesel are expected to change from source to source, so the toxicity of diesel fuels is expected to be variable. The following sections summarize the toxicity of diesel fuel to plants, aquatic organisms, birds, and mammals.

3.3.1.1 Plants. Petroleum released to the aquatic environment is expected to be toxic to aquatic plants. Toxicity tests have shown that the water-soluble components of petroleum are toxic to an algal species (*Chlorella vulgaris*) (Kauss and Hutchinson 1975). However, in this

specific study, the toxicity was short term. The algal community recovered after a "lag phase". It was theorized (Kauss and Hutchinson 1975) that this trend was due to the loss of highly volatile fractions from the testing chamber over time. Exposure to water extracts of No. 2 Fuel Oil depressed algal biomass in communities and resulted in blue-green algal dominance and decreased diatom occurrence (Bott and Rogenmuser 1978).

3.3.1.2 Aquatic Organisms. Moles et al. (1979) tested the acute toxicity of Prudhoe Bay crude oil to several Alaskan freshwater and anadromous fish. Salmonids were the most sensitive species tested, and demonstrated median tolerance limits (the concentration at which one half the organisms survive in 96 hours, the same as LC_{50} [lethal concentration for 50 percent of the organisms]) ranging from 2.7 to 4.4 mg/L. The three-spined stickleback was more tolerant, with an LC_{50} of 10.4 mg/L. Klein and Jenkins (1983) studied the toxicity of the water soluble fraction of jet fuel to fish. Growth of fry was retarded by 1.5 mg/L of the water soluble fraction of JP-8 (jet fuel with de-icer). In a study conducted by Hedtke and Puglisis (1982), the method of introducing the oil to the test chamber was an important variable driving toxicity. Emulsified oils were substantially more toxic than either floating oils or the water soluble fraction. The 96-hour LC_{50} for fathead minnows (*Pimephales promelas*) exposed to the emulsion of No. 2 jet fuel was 38.6 mg/L (concentration used to calculate the TRV).

Aquatic organisms other than fish may also be exposed to diesel fuel in the environment. Studies have shown that freshwater arctic zooplankton may be more sensitive to oil pollution than any other arctic freshwater organisms (O'Brien 1978). Geiger and Buikema (1981) estimated an LC_{20} (concentration lethal to 20 percent of the test organisms) of No. 2 Fuel oil to *Daphnia pulex* of 5.6 mg/L (concentration used to calculate TRV).

3.3.1.3 Birds. Petroleum hydrocarbons in the environment may affect bird reproduction. External application of Number 2 fuel oil to mallard (*Anas platyrhynchos*) and common eider (*Somateria mollissima*) eggs significantly increased embryo mortality (Albers 1977; Szaro and Albers 1977). Mallard eggs were treated with 1, 5, 10, 20, and 50 μl of fuel oil. Ingestion of crude oil by mallard ducks at a concentration of five percent by weight in the diet resulted in depressed growth (Szaro et al. 1978). Hartung (1964) demonstrated a decrease in weight gain in mallard ducks during the first 10 days after receiving 6,000 mg/kg No. 2 fuel oil (concentration used to calculate TRV). However, after 34 days, there was no difference between treatment groups and the controls.

3.3.1.4 Mammals. The available literature does not present a great deal of information regarding the toxicity of diesel fuel to mammals, but it can be represented by the toxicity of the compound to rats. Diesel fuel is slightly toxic to rats based on an acute oral LD_{50} (lethal dose for 50 percent of the organisms) of 7,380 mg/kg (Beck et al. 1982) (dose used to calculate TRV). A dermal LD_{50} in rabbits was reported as >4,290 mg/kg (Beck et al. 1982).

3.3.2 Ethylbenzene

Ethylbenzene is a COC in soil/sediment at the Wainwright installation. It is a VOC, and most toxicity information in the literature relates to its inhalation. A summary of the relevant toxicity information is presented below.

3.3.2.1 Plants. In a cell multiplication inhibition test using *Microcystis aeruginosa* (algae) the toxicity threshold of ethylbenzene was 33,000 µg/L. The toxicity threshold of *Scenedesmus quadricauda* (green algae) to ethylbenzene was >160,000 µg/L (Verschueren 1983). Galassi et al. (1988 in AQUIRE 1994) reported an EC_{50} (effective concentration for 50 percent of the organisms) (growth) of 4,600 µg/L for *Selenastrum capricornutum* (green algae).

3.3.2.2 Aquatic Organisms. Ethylbenzene was a COC in soil/sediment but not in water. As a result, no quantitative presentation of water exposure was conducted in this ERA.

3.3.2.3 Birds. There is no information in the literature regarding the toxicity of ethylbenzene to birds.

3.3.2.4 Mammals. One study regarding the toxicity of ethylbenzene administered orally to rats reported an LD_{50} 5,460 mg/kg (Budavari 1989) (dose used to calculate TRV).

3.3.3 Xylene

Xylene is a COC in soil/sediment at the Wainwright installation. It is a VOC, and most toxicity information in the literature relates to the inhalation of xylene. A summary of the relevant information is presented below.

3.3.3.1 Plants. In a study of the green alga, *Selenastrum capricornutum*, xylene decreased growth at concentrations of 72,000 µg/L (Gaur 1988 in AQUIRE).

3.3.3.2 Aquatic Organisms. Xylene is not a COC in surface water, so the toxicity to aquatic organisms is not presented.

3.3.3.3 Birds. When mallard eggs were immersed in xylene (1 percent and 10 percent) for 30 seconds, there were no significant effects at concentrations of 10 percent on embryonic weight & length when compared to controls [Hoffman and Eastin 1981 in Hazardous Substance Data Bank (HSDB) 1994]. Japanese quail (*Coturnix japonica*) fed xylene demonstrated no sign of toxicity up to 5,000 ppm (USFWS 1986). The LC_{50} was >20,000 ppm (USFWS 1986). Hill and Camardese (1986) report a maximum dietary exposure level for Japanese quail of 625 mg/kg total xylenes (dose used to calculate TRV).

3.3.3.4 Mammals. Ingestion of xylene in mammals may cause prenatal mortality, growth inhibition, and malformations, primarily cleft palate. The LD_{50} for ingestion of xylene (rat) was reported as 4,300 mg/kg (Clayton and Clayton 1981) (dose used to calculate TRV).

3.3.4 Tetrachloroethene

Tetrachloroethene is a COC in soil/sediment at the Wainwright installation. It is a halogenated hydrocarbon (Cl_2CCl_2) used in commercial drycleaning operations and as a solvent. It has demonstrated experimental carcinogenic, teratogenic, and reproductive effects (Sax and Lewis 1989). Toxicity information for this compound is limited. Available information is summarized below.

3.3.4.1 Plants. Specific toxicity information for plants and tetrachloroethene is not available.

3.3.4.2 Aquatic Organisms. Tetrachloroethene was not found to be a COC in water, so it is not quantitatively evaluated in the risk assessment for aquatic organisms.

3.3.4.3 Birds. No toxicity information relating birds to tetrachloroethene was available.

3.3.4.4 Mammals. Buben and O'Flaherty (1985 in Opresko et al. 1994) conducted a six-week subchronic exposure test of tetrachloroethene on mice. Seven dose levels were administered by gavage five days per week. Hepatotoxicity was noted at 100 mg/kg/d or greater doses. A subchronic NOAEL was determined to be 20 mg/kg/d. Applying conversion and uncertainty factors specified in Opresko et al. (1994), a final NOAEL of 1.4 mg/kg/d was derived. This is the value used to calculate the TRV.

3.3.5 Trimethylbenzene

Trimethylbenzene is a COC in soil/sediment, but not in surface water. Trimethylbenzene exists in three isomeric forms, 1,2,3-trimethylbenzene, 1,2,4-trimethylbenzene, and 1,3,5-trimethylbenzene. The exposure concentration for trimethylbenzene is based on the sum of the averages of the 1,2,4- and 1,3,5- isomers.

3.3.5.1 Plants. Specific toxicity information for plants and trimethylbenzene is not available.

3.3.5.2 Aquatic Organisms. Trimethylbenzene was not found to be a COC in water, so it is not quantitatively evaluated in the risk assessment for aquatic organisms.

3.3.5.3 Birds. No toxicity information relating birds to trimethylbenzene was available.

3.3.5.4 Mammals. No toxicity information relating mammals to trimethylbenzene was available.

3.3.6 Naphthalene

Naphthalene was determined to be a COC in soil/sediment, but not in surface water. Naphthalene belongs to the group of chemical compounds known as PAHs. PAHs consist of hydrogen and carbon atoms combined to form two or more fused benzene rings (Eisler 1987). The structure of naphthalene is a two-ring, unsubstituted molecule ($C_{10}H_8$). Specific toxicity data for naphthalene is generally lacking, so the following discussion may include toxicity information for other PAH compounds.

3.3.6.1 Plants. Specific toxicity information for plants exposed to naphthalene or other PAHs is not available. Some general trends have been observed by researchers (EPA 1980; Lee and Grant 1981; Wang and Meresz 1982; Edwards 1983; Sims and Overcash 1983 in Eisler 1987). PAHs may be absorbed from soil through plant roots and can be translocated to other

parts of the plant. The factors that appear to govern plant uptake include soil concentration, water solubility, and soil type.

3.3.6.2 Aquatic Organisms. Naphthalene was not found to be a COC in water so it is not quantitatively evaluated in terms of exposure of aquatic organisms.

3.3.6.3 Birds. There is limited information regarding the toxicity of PAHs to birds. In a study conducted by Patton and Dieter (1980), mallards fed 4,000 mg PAHs/kg for 7 months demonstrated increased liver weight and blood flow to the liver. The PAH mixture tested contained naphthalenes, naphthenes, and phenanthrene.

3.3.6.4 Mammals. Some PAHs are animal carcinogens. However, unsubstituted PAHs with fewer than four rings (as is naphthalene) have not been shown to induce tumorigenic activity (Eisler 1987). In addition, although unsubstituted PAHs are highly lipid soluble, they do not accumulate in mammalian tissue because of ready metabolization by animals (EPA 1980; Lee and Grant 1981). Specific studies regarding the toxicity of naphthalene to mammals are limited. 1,780 mg/kg body weight of naphthalene caused acute oral toxicity in rats (Sims and Overcash 1983 in Eisler 1987). The HSDB (1994) reports a NOAEL dose of 50 mg/kg naphthalene (dose used to calculate TRV) for a laboratory rat.

3.3.7 Aluminum

Aluminum was found to be a COC in surface water, but not in soils/sediments. Aluminum is an ubiquitous naturally-occurring element. It is amphoteric, with solubility lowest at a pH of 5.5 and increasing as pH deviates from 5.5 in either direction (EPA 1988). Attempts to relate toxicity to pH have yielded diverse results. Some researchers have reported a direct correlation between increased aluminum toxicity and increased pH (Freeman and Everhardt 1971; Hunter et al. 1980 in EPA 1988), although other researchers have reported decreased toxicity (Call 1984; Boyd 1979; Kimball unpublished in EPA 1988).

3.3.7.1 Plants. According to the EPA (1988), single-celled plants are more sensitive to aluminum exposure than other plants. Concentrations of 810 µg/L inhibited the growth of the diatom *Cyclotella meneghiniana* (Rao and Subramanian 1982 in EPA 1988). The green alga, *Selenastrum capricornutum* (Call 1984 in EPA 1988) was affected by concentrations of aluminum of 460 µg/L. The Eurasian watermilfoil demonstrated decreased root weight when exposed to aluminum concentrations of 2,500 µg/L. Concentrations of aluminum of 8,000 µg/L in culture solutions resulted in toxic threshold concentrations in rice shoots and soybean leaves of 20 and 30 mg/kg, respectively (Wallace and Romney 1977 in Gough et al. 1979). Most of the aluminum remained in the roots of the plant.

3.3.7.2 Aquatic Organisms. Aluminum is toxic to carp (*Cyprinus carpio*) at 4,000 µg/L (48-hr LC_{50}) (Muramoto 1981 in EPA 1988). A 96-hr LC_{50} of 3,600 µg/L was reported for the brook trout (*Salvelinus fontinalis*) (Decker and Menendez 1974 in EPA 1988). TRVs for the nine-spined stickleback are based on this value. In a chronic toxicity test with *Daphnia magna*, a reduction in survival (29 percent) was shown to occur at concentrations of 1,020 µg/L (Kimball

unpublished manuscript in EPA 1988). The organisms that survived this test did not exhibit any adverse effect on reproduction.

3.3.7.3 Birds. In a toxicity study conducted using red-winged blackbirds as the test organism, aluminum concentrations >111 mg/kg body weight were toxic (LD_{50}) (Schafer et al. 1983). In a study conducted by Cakir et al. (1978 in NAS 1980), a no-effect concentration of 486 ppm aluminum was reported for both turkeys and chicks. A 4-month reproductive study using ringed doves determined a chronic NOAEL of 111.4 mg/kg/d (Carrier et al. 1986 in Opresko et al. 1994). This is the dose used to calculate the TRV.

3.3.7.4 Mammals. No toxicity study using wild mammalian species was found in the literature. Calves and sheep fed 1,200 and 1,215 ppm (respectively) of aluminum in the diet showed no adverse effects (Valdivia et al. 1978; Bailey 1977 in NAS 1980). The value reported by Bailey (1977 in NAS 1980) is used to calculated the TRV for caribou. Studies reported in ATSDR (1990a) indicate an acute systemic NOAEL for rats exposed to aluminum of 108 mg/kg-bw/day (dose used to calculate TRV for brown lemming and arctic fox).

3.3.8 Cadmium

Cadmium was determined to be a COC in soils/sediments. It was detected above action levels at one location at a concentration of 72 mg/kg.

3.3.8.1 Plants. Cadmium in soil is absorbed passively by plants and translocated freely within the plant. Its phytotoxicity is related to alteration of cell membrane permeability, and at least some toxic effects are linked specifically to interference of zinc-dependent uptake and translocation processes (Foy et al. 1978). Chlorosis is one of the general symptoms of cadmium toxicity in plants and appears to be caused by direct or indirect interaction of cadmium with foliar iron (Foy et al. 1978). Allaway (1968) noted that 3 mg/kg cadmium in the tissues of plants depressed growth. Traynor and Knezek (1973) reported that corn grown on cadmium-enriched soils readily absorbed and translocated the element. They also found growth reduction in corn at its maximum when 281 mg/kg cadmium was added to soil resulting in a plant concentration of 131 mg/kg (ash weight basis). Cadmium has been found to concentrate in plants to as high as ten times the soil concentration (Chaney and Hornick 1977 in Kabata-Pendias and Pendias 1984). Cadmium toxicity to soybeans was described as a 10 percent reduction in yield and discoloration of the plants at soil concentrations as low as 2.5 mg/kg. This soil concentration was also attributed to 21 percent and 40 percent yield reductions in wheat and lettuce, respectively (Haghiri 1973). Levels of 3 to 8 mg/kg (mean of four studies - 5.25 mg/kg) were reported as phytotoxic by Kebata-Pendias and Pendias (1984).

3.3.8.2 Aquatic Organisms. Cadmium was a COC in soil/sediment, but not in surface water. As a result, no aquatic toxicity values are presented.

3.3.8.3 Birds. In a study by DiGiulio and Scanlon (1984), mallards (*Anas platyrhynchos*) were chronically exposed to cadmium-spiked food. They found significant effects on energy metabolism at 450 ppm, but not at 150 ppm. The 450 ppm group also had reduced body and liver weights. White and Finley (1978 in Eisler 1985) reported no loss in body weight in adult

mallards fed 200 ppm cadmium for 90 days. Mallard ducklings showed mild to severe kidney lesions when fed 20 ppm dietary cadmium for 12 weeks (Cain et al. 1983 in Eisler 1985). After eight weeks the birds showed reduced hemoglobin concentrations and packed cell volume, and increased serum glutamic pyruvic transaminase. Japanese quail (*Coturnix japonica*) given 75 ppm cadmium for six weeks had anemia, bone marrow hypoplasia (abnormal development), and heart damage (Eisler 1985). Reduced body weight occurs in poultry at 400 ppm, and egg fertility and hatchability are adversely affected at 100 ppm (Puls 1988). Puls (1988) reported that poultry exhibited nephritis at 3 ppm per 1 ppm in the diet (Lehman 1954). Puls (1988) reported that poultry exhibited nephritis at 3 pm dietary cadmium, but egg production was increased. At 8 to 60 ppm dietary cadmium reduced food consumption, reduced egg production, and thinned eggshells in poultry (Puls 1988). The TRV in this study is based on a 3 ppm concentration NOAEL presented by Leach et al. (1979 in NAS 1980). This is equivalent to a dose of 0.53 mg/kg-body weight, based on a dietary conversion factor of 0.175 mg/kg-body weight per 1 ppm in the diet (Sax and Lewis 1989).

3.3.8.4 Mammals. Mammals have no effective mechanism for the elimination of ingested cadmium; therefore, with time, the cadmium tends to accumulate in the liver and kidney. It tends to be very persistent in the kidney and can cause renal tubular damage (NAS 1980). The low lethal oral doses in rats and guinea pigs range from 150 to 250 mg/kg (Eisler 1985). Doyle et al. (1972) found that 30 to 60 ppm cadmium in the diet of sheep for 191 days reduced growth and food intake. In a 30-month study with rats, elevated blood pressure occurred at the lowest level tested (1 ppm) (Perry et al. 1977 in NAS 1980). The maximum tolerable dietary cadmium level recommended by NAS (1980) for domestic mammals and poultry is 0.5 ppm. This is the TRV used for caribou. Schroeder and Mitchner (1971 in Opresko et al. 1994) reported a LOAEL of 10 ppm cadmium (in water) and 0.1 ppm (in food). Opresko et al. derived a NOAEL of 0.2 mg/kg/day, which is the value used as the TRV for brown lemming and arctic fox.

3.3.9 Iron

Iron is an essential trace element required by both plants and animals. It is a COC in surface water and soil/sediment at the Wainwright installation.

3.3.9.1 Plants. In a study conducted by Foy et al. (1978), concentrations of 100 to 500 ppm soluble iron in soil were toxic to rice.

3.3.9.2 Aquatic Organisms. Iron may be a threat in aquatic environments in the form of precipitates that can destroy habitat, coat gills, and inhibit oxygen uptake. The EPA uses 1,000 μg/L as the chronic AWQC protective of aquatic life (dose used to calculate TRV) (EPA 1986c). In a study conducted by Warnick and Bell (1969 in EPA 1976) mayflies, stoneflies, and caddisflies were affected by iron concentrations of 320 μg/L (96-hr LC_{50}). Doudoroff and Katz (1953 in EPA 1976) found iron concentrations of 1,000 to 2,000 μg/L toxic to *Esox lucius* (northern pike) and trout (species not reported).

3.3.9.3 Birds. There are few studies available that address the toxicity of iron to species of wild birds. There were no adverse effects produced in turkeys at concentrations of 440 ppm (Woerpel and Balloun 1964 in NAS 1980). NAS (1980) recommends that the maximum tolerable

level of dietary iron of 1,000 ppm be used for poultry. The 1,000 ppm dose converts to 70.0 mg/kg for a maximum tolerable dietary level for a chicken (dose used to calculate TRV).

3.3.9.4 Mammals. At high concentrations, iron is toxic to livestock and interferes with phosphorus metabolism (NAS 1974 in EPA 1976). Cattle fed 477 µg/g iron demonstrated a slight decrease in weight gain; concentrations of 1,677 µg/g of iron produced a significant decline in growth rate (EPA 1985). Shanas and Boyd (1969 in NAS 1980) report an acute LD_{50} dose of iron for the rat to be 1,000 mg/kg (dose used to calculate TRV for brown lemming and arctic fox). The maximum tolerable dietary level of 500 ppm of iron for sheep was used to calculate the TRV for caribou (NAS 1980).

3.3.10 Lead

Lead was found to be a COC in soils/sediments, but not in surface water. Lead is a trace element naturally found in environmental media (e.g., soil, water, etc.); however, it is neither essential nor beneficial to living organisms (Eisler 1988).

3.3.10.1 Plants. Lead inhibits plant growth, reduces photosynthesis, and reduces mitosis and water absorption (Eisler 1988). Concentrations of 500 mg/kg in soils were found to result in reduced pollen germination in several weed species, but the same study found that 46 mg/kg lead concentrations in soil did not have adverse effects on pollen germination (USACOE 1991).

3.3.10.2 Aquatic Organisms. Lead was not found to be a COC in surface water. As a result, aquatic organisms are not evaluated quantitatively for lead exposure in the ERA.

3.3.10.3 Birds. The bulk of the toxicity information in the literature regarding avian exposure to lead concerns waterfowl that have ingested spent lead shot and died. These results are reported as body burdens of lead. There is, however, limited dose-response information available for some species. Mautino and Bell (1987) reported neurological effects in mallard ducks that had ingested and absorbed lead shot for a total intake of 423.8 mg/kg body weight. Young American kestrels (*Falco sparverius*) (1 day old) that ingested 125 and 625 mg/kg body weight of lead, showed significantly depressed growth and hematocrit values (Hoffman et al. 1985). Damron et al. (1969 in NAS 1980) report a NOAEL of 100 ppm for lead in a 28-day toxicity study using 4-week old chickens as the test organism. This is the dose used to calculate the TRV.

3.3.10.4 Mammals. Lead may affect the survival, growth, development, and metabolism of animal species. Rats are affected by 5 to 108 mg/kg body weight (acute oral dose); dogs by 0.32 mg/kg body weight daily (chronic oral dose); and horses by chronic dietary concentrations of 1.7 mg/kg (Eisler 1988). Azar et al. (1973 in Opresko et al. 1994) reports an oral dose of 8.0 mg/kg-bw/day to be a chronic NOAEL for laboratory rats (dose used to calculate TRV for brown lemming and arctic fox). Fick et al. (1976 in NAS 1980) report a chronic NOAEL for sheep of 10 ppm in diet. This value was used to calculate the TRV for caribou.

3.3.11 Manganese

Manganese was determined to be a COC in surface water and soil/sediment. Manganese is considered to be an essential nutrient for animals (ATSDR 1990b), and it is important for growth and reproduction. The toxicity of manganese can be affected by pH and water hardness.

3.3.11.1 Plants. In a four-day study conducted using duckweed (*Lemna minor*), an EC_{50} (reduction in growth) was reported of 31,000 µg/L (Wang 1986 in AQUIRE 1990). Lewis et al. (1979) studied the species composition of freshwater phytoplankton populations when exposed to manganese. Population composition was altered at 0.1 mg/L manganese. Soil concentrations of 1,500 to 3,000 mg/kg reported as phytotoxic to all plant species (Kabata-Pendias and Pendias 1984).

3.3.11.2 Aquatic Organisms. In a study conducted by Doudoroff and Katz (1953), brook trout were killed within 24 hours when exposed to manganese concentrations of 6,250 µg/L. Rainbow trout have a reported LC_{50} of 2,910 µg/L (Pickering et al. 1983) (dose used to calculate TRV for nine-spined stickleback). *Daphnia* spp. have a reported 16 percent reproductive impairment in water with concentrations of 4,100 µg/L (Biesinger and Christensen 1972 in Lewis et al. 1979) (dose used to calculate TRV for *Daphnia* spp).

3.3.11.3 Birds. Vohra and Kratzer (1968 in NAS 1980) exposed young turkeys to dietary manganese for 21 days. A no observed effect level (NOEL) of 4,080 ppm was derived. A NOAEL of 1,000 ppm of manganese was reported for young chicken in a 20-week diet study (Gallup and Norris 1939 in NAS 1980). This is the value used to calculate the avian TRV.

3.3.11.4 Mammals. When fed 9,000 ppm manganese, sheep demonstrated reduced feed intake (Puls 1988). NAS recommends maximum tolerable levels of 1,000 ppm for cattle (15 mg/kg body weight) and sheep (40 mg/kg body weight). The value for sheep was used to calculate the TRV for caribou. A NOAEL of 930 mg/kg-bw/day is reported for rats in ATSDR (1990b). The TRVs for brown lemming and arctic fox are based on this dose.

3.3.12 Vanadium

Vanadium was determined to be a COC in surface water. It was detected above action levels at one location at a concentration of 31 µg/L. Vanadium is amphoteric in aqueous solution, and as the pH of the solution is varied, soluble vanadium occurs in different ionic species (Lee 1983). In fresh water, vanadium generally exists in solution as the vanadyl ion (V^{4+}) under reducing conditions and the vanadate ion (V^{5+}) under oxidizing conditions, or absorbed onto particulate matter (Wehrli and Stumm 1989 in ATSDR 1991).

3.3.12.1 Plants. In general, marine plants contain higher levels of vanadium than terrestrial plants (Lee 1983). In the terrestrial environment, bioconcentration is more commonly observed among the lower plant phyla (ATSDR 1991). The vanadium levels in terrestrial plants are dependent upon the amount of water-soluble vanadium available in the soil, pH, and growing conditions, although it has been found that the uptake is low (Byerrum et al. 1974 in ATSDR 1991). Vanadium influences the biochemical processes in plants. It appears to play a role in the

central processes regulating growth of algae with about 20 ppb vanadium stimulating growth (Lee 1983). Vanadium has been identified as an essential micronutrient, and levels of less than 10 ppm are found in phytoplankton and attached algae (Lee 1983). High concentrations, however, have a toxic effect on algae. The toxic threshold for vanadium content in algae was determined to be 150 - 200 mg/g dry weight. The growth of *Ceratium hirundinella* (diatom) was inhibited by 100 ppb vanadium (Bruno and McLaughlin 1977 in Lee 1983).

3.3.12.2 Aquatic Organisms. Very few vanadium toxicity tests have been conducted on invertebrates. An LC_{50} for *Daphnia* spp. of 1,520 was used to derive the TRV (Suter and Mabrey 1994). The 96-hour LC_{50} ranged from 4.8 to 55 ppm vanadium (as $VOSO_4$ or V_2O_5) for fathead minnow and bluegill (Tarzwell and Henderson 1956, 1960 in Lee 1983). Vanadium was moderately toxic to juvenile rainbow trout (*Salmo gairdneri*) and white fish (*Coregonus clupeaformis*) (96-hour LC_{50} of 6.4 and 17.4 ppm, respectively), with toxicity increasing slightly with decreasing pH (Giles and Klaverkamp 1982 in Lee 1983). The LC_{50} of 6.4 ppm for rainbow trout was used to derive the TRV for the nine-spined stickleback.

3.3.12.3 Birds. There are few studies available that address the toxicity of vanadium to species of wild birds. Day-old chicks exposed to 8 to 10 ppm dietary vanadium showed evidence of reduced growth (NAS 1980). The value of 10 ppm was used to derive the TRV for Lapland longspur, brant, glaucous gill, and pectoral sandpiper. Other studies with young chicks indicate a tolerance of 20 to 25 ppm vanadium; however, 30 ppm fed to laying hens reduced egg quality and 35 ppm was toxic for chick growth (NAS 1980).

3.3.12.4 Mammals. At low levels, vanadium has been shown to be essential for normal growth and proper physiological function in livestock. Calves fed vanadium in the diet (20 mg/kg of body weight) manifested diarrhea, emaciation, and prostration within three days (NAS 1980). Sheep showed a 65 percent death rate within 80 hours when given 40 mg vanadium per kg body weight as NH_4VO_3. Lambs fed 100 to 200 ppm vanadium showed no signs of effects on growth rate, but a significant increase in tissue levels was observed (NAS 1980). Weanling rats tolerated 20 ppm vanadium (as sodium metavanadate), but 40 ppm reduced growth. NAS (1980) reported a maximum tolerable level of 50 ppm for cattle and sheep, which was used to calculate the TRV for caribou. An NOAEL of 0.21 mg/kg/day in rats was used to calculate the TRV for brown lemming and arctic fox (Domingo et al. in Opresko et al. 1994).

3.3.13 Zinc

Zinc was determined be a COC in both surface water and soils/sediments. Zinc is considered to be an essential nutrient for animals (Eisler 1993) and is necessary for plant growth. Deficiencies of zinc in the diet may retard growth in animals (Eisler 1993).

3.3.13.1 Plants. According to information presented in Eisler (1993), plants that are sensitive to zinc concentrations may die when soil levels are in excess of 100 mg/kg or when plant zinc content exceeds 178 mg/kg (dry weight). The amount of zinc absorbed from soil in plants is dependent upon soil-specific characteristics. USACOE (1991) reports that several species of plants find average concentrations of zinc of 270 mg/kg in soil to be phytotoxic.

3.3.13.2 Aquatic Organisms. Nehring and Goettl (1974) evaluated the toxicity of zinc to four trout species and reported 14-day LC_{50}s of 410 µg/L for rainbow trout (*Oncorhynchus mykiss*) (hardness = 20 - 51 mg/L); 640 µg/L for brown trout (*Salmo trutta*) (hardness = 22 to 55 mg/L); 670 µg/L for cutthroat trout (*Salmo clarki*) (hardness = 22 to 58 mg/L); and 960 µg/L for brook trout (*Salvelinus fontinalis*) (hardness not measured). The TRV for the nine-spined stickleback was based on a NOAEL of 140 µg/L for the rainbow trout (EPA 1987). The TRV for *Daphnia* spp. was based on a NOAEL of 47.3 µg/L (EPA 1987).

3.3.13.3 Birds. When ducks were fed 2,500 to 3,000 mg/kg ration of zinc, or alternately, force-fed zinc at 742 mg zinc/kg body weight, survival was reduced (Eisler 1993). Chickens were more resistant to zinc exposure; 8,000 mg zinc/kg ration was lethal to chicks (Eisler 1993). Elevated levels of zinc (20 g zinc/kg ration) are given to poultry to induce molting and subsequently reduce egg deposition (Eisler 1993). A four-week study conducted by Roberson and Schaible (1960 in NAS 1980) calculated a NOAEL of 1,000 ppm for 1-day-old chicks. This value was used to calculate the avian TRV.

3.3.13.4 Mammals. According to Eisler (1993), zinc is relatively non-toxic to mammals (as would be expected for an essential trace element). There is a large range in concentrations between normal dietary intakes and those concentrations expected to cause harm. Adult male rats, when fed zinc at levels of 500 mg/kg diet, were adversely affected; spermatogenesis was arrested, and testes enlarged (Eisler 1993). Zinc concentrations of 6,820 mg zinc/kg ration suppressed rat growth and produced changes in the pancreas (Eisler 1993). A chronic NOAEL for laboratory rats of 160 mg/kg-bw/day is reported by Schlicker and Cox (1968 in Opresko et al. 1994). The TRV for the brown lemming and arctic fox was derived from this toxicity value. During a 10 week study conducted by Ott et al. (1966 in NAS 1980), a NOAEL of 500 mg/kg for sheep was determined. This value was used to calculate the TRV for caribou.

3.3.14 Characterization of Effects

In this section toxicity information is presented for representative ecological receptors that will be evaluated in the risk characterization section of this report (Section 3.4). Potential impacts to aquatic receptors are evaluated by comparing exposure concentrations to TRVs. Potential impacts to terrestrial wildlife are evaluated for the representative species based on comparisons of estimated exposures to TRVs. TRVs for the representative aquatic species are presented in Table 3-15. Exposure to COCs for the representative species is primarily through diet, which may include plants, fish, and aquatic invertebrates, soils, and surface water. TRVs are derived for COCs in surface water and soil/sediment. TRVs for the representative bird species are presented in Tables 3-16 and 3-17, and for the representative mammal species in Tables 3-18 and 3-19.

3.3.14.1 Toxicity Reference Values. TRVs were derived by selecting toxicity values from the literature and then extrapolating to the species of concern. Uncertainty factors (UF) and body scaling factors were used in the extrapolation process as described below. Tables 3-15 to 3-19 present the TRVs.

TABLE 3-15. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF AQUATIC ORGANISMS AT THE WAINWRIGHT INSTALLATION (METALS)

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	CONCENTRATION ($\mu g/L$)	TEST SPECIES	NOAEL UF	INTERSPECIES UF	PROTECTED SPECIES UF	TRV (mg/L)	REFERENCE
Aluminum	nine-spined stickleback	96 hour - LC_{50}	3,600	brook trout	20	2	1	92	Decker and Menendez 1974 in EPA 1988
	Daphnia spp.	chronic reproductive impairment LOAEL	1,020	*D. magna*	10	1	1	102	Kimball manuscript in EPA 1988
Iron	nine-spined stickleback	EPA chronic water quality criteria	1,000	all aquatic life	1	1	1	1,000	EPA 1986c
	Daphnia spp.	EPA chronic water quality criteria	1,000	all aquatic life	1	1	1	1,000	EPA 1986c
Manganese	nine-spined stickleback	28 day - LC_{50}	2,910	rainbow trout	20	2	1	73	Pickering et.al. 1983
	Daphnia spp.	chronic reproductive impairment LOAEL	4,100	*Daphnia* spp.	10	1	1	410	Beisinger and Christensen 1972 in Lewis et al. 1979
Vanadium	nine-spined stickleback	96 hours - LC_{50}	6,400	rainbow trout	20	2	1	160	Giles and Klaverdamp 1982 in Lee 1983
	Daphnia spp.	LC_{50}	1,520	*Daphnia* spp.	20	1	1	76	Kimball (no date) in Suter and Mabrey 1994
Zinc	nine-spined stickleback	chronic lifecycle NOAEL	140	rainbow trout	1	2	1	70	EPA 1987 in Eisler 1993
	Daphnia spp.	chronic lifecycle NOAEL at 104 mg/L $CaCO_3$	47.3	*D. magna*	1	1	1	47	Chapman et al. manuscript in EPA 1987

TABLE 3-16. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF BIRDS AT THE WAINWRIGHT INSTALLATION (METALS)

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Aluminum	Lapland longspur	chronic NOAEL; four month reproductive study	111.4	ringed dove	1	0.56	2	2	50	Carriere et al. 1986 in Opresko et al. 1994
	brant	chronic NOAEL; four month reproductive study	111.4	ringed dove	1	2.03	2	2	14	Carriere et al. 1986 in Opresko et al. 1994
	glaucous gull	chronic NOAEL; four month reproductive study	111.4	ringed dove	1	2.10	2	2	13	Carriere et al. 1986 in Opresko et al. 1994
	pectoral sandpiper	chronic NOAEL; four month reproductive study	111.4	ringed dove	1	0.80	2	2	35	Carriere et al. 1986 in Opresko et al. 1994
Cadmium	Lapland longspur	chronic NOAEL; 48 week uptake and reproductive study	0.53	chicken	1	0.32	2	2	0.41	Leach et al. 1979 in NAS 1980
	brant	chronic NOAEL; 48 week uptake and reproductive study	0.53	chicken	1	1.18	2	2	0.11	Leach et al. 1979 in NAS 1980
***	glaucous gull	chronic NOAEL; 48 week uptake and reproductive study	0.53	chicken	1	1.22	2	2	0.11	Leach et al. 1979 in NAS 1980
	pectoral sandpiper	chronic NOAEL; 48 week uptake and reproductive study	0.53	chicken	1	0.46	2	2	0.29	Leach et al. 1979 in NAS 1980
Iron	Lapland longspur	NOAEL; 28 day growth study	70.0	chicken	1	0.32	2	2	55	McGhee et al. 1965 in NAS 1980
	brant	NOAEL; 28 day growth study	70.0	chicken	1	1.18	2	2	15	McGhee et al. 1965 in NAS 1980
	glaucous gull	NOAEL; 28 day growth study	70.0	chicken	1	1.22	2	2	14	McGhee et al. 1965 [illegible]S 1980

TABLE 3-16. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF BIRDS AT THE WAINWRIGHT INSTALLATION (METALS) (CONTINUED)

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Iron (Continued)	pectoral sandpiper	NOAEL; 28 day growth study	70.0	chicken	1	0.46	2	2	38	McGhee et al. 1965 in NAS 1980
Lead	Lapland longspur	NOAEL; 4 week old chicks 28 day study	17.5	chicken	1	0.32	2	2	14	Damron et al. 1969 in NAS 1980
	brant	NOAEL; 4 week old chicks 28 day study	17.5	chicken	1	1.18	2	2	3.7	Damron et al. 1969 in NAS 1980
	glaucous gull	NOAEL; 4 week old chicks 28 day study	17.5	chicken	1	1.22	2	2	3.5	Damron et al. 1969 in NAS 1980
	pectoral sandpiper	NOAEL; 4 week old chicks 28 day study	17.5	chicken	1	0.46	2	2	9.5	Damron et al. 1969 in NAS 1980
Manganese	Lapland longspur	chronic NOAEL; 140 day mortality study	175	chicken	1	0.32	2	2	140	Gallup and Norris 1939 in NAS 1980
	brant	chronic NOAEL; 140 day mortality study	175	chicken	1	1.18	2	2	37	Gallup and Norris 1939 in NAS 1980
	glaucous gull	chronic NOAEL; 140 day mortality study	175	chicken	1	1.22	2	2	36	Gallup and Norris 1939 in NAS 1980
	pectoral sandpiper	chronic NOAEL; 140 day mortality study	175	chicken	1	0.46	2	2	95	Gallup and Norris 1939 in NAS 1980
Vanadium	lapland longspur	NOAEL; maximum tolerable dietary level	1.8	poultry	1	0.32	2	2	1.4	NAS 1980
	brant	NOAEL; maximum tolerable dietary level	1.8	poultry	1	1.18	2	2	0.38	NAS 1980

TABLE 3-16. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF BIRDS AT THE WAINWRIGHT INSTALLATION (METALS) (CONTINUED)

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Vanadium (Continued)	glaucous gull	NOAEL; maximum tolerable dietary level	1.8	poultry	1	1.22	2	2	0.37	NAS 1980
	pectoral sandpiper	NOAEL; maximum tolerable dietary level	1.8	poultry	1	0.46	2	2	0.98	NAS 1980
Zinc	Lapland longspur	subchronic NOAEL; four week dietary study	175	chicken	10	0.32	2	2	14	Roberson and Schaible 1960 in NAS 1980
	brant	subchronic NOAEL four week dietary study	175	chicken	10	1.18	2	2	3.7	Roberson and Schaible 1960 in NAS 1980
	glaucous gull	subchronic NOAEL four week dietary study	175	chicken	10	1.22	2	2	3.6	Roberson and Schaible 1960 in NAS 1980
	pectoral sandpiper	subchronic NOAEL four week dietary study	175	chicken	10	0.46	2	2	9.5	Roberson and Schaible 1960 in NAS 1980

TABLE 3-17. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF BIRDS AT THE WAINWRIGHT INSTALLATION (ORGANICS)

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
DRPH	Lapland longspur	decreased weight gain LOAEL	6,000	mallard	10	0.29	2	2	520	Hartung 1964
	brant	decreased weight gain LOAEL	6,000	mallard	10	1.07	2	2	140	Hartung 1964
	glaucous gull	decreased weight gain LOAEL	6,000	mallard	10	1.10	2	2	140	Hartung 1964
	pectoral sandpiper	decreased weight gain LOAEL	6,000	mallard	10	0.42	2	2	360	Hartung 1964
Ethylbenzene	Lapland longspur	NA								
	brant	NA								
	glaucous gull	NA								
	pectoral sandpiper	NA								
Xylene	Lapland longspur	Maximum dietary exposure	608	Japanese quail	10	0.60	2	2	26	Hill and Camardese 1986
	brant	Maximum dietary exposure	608	Japanese quail	10	2.16	2	2	7.2	Hill and Camardese 1986
	glaucous gull	Maximum dietary exposure	608	Japanese quail	10	2.23	2	2	6.8	Hill and Camardese 1986
	pectoral sandpiper	Maximum dietary exposure	608	Japanese quail	10	0.85	2	2	18	Hill and Camardese 1986
Tetrachloroethene	Lapland longspur	NA								
	brant	NA								
	glaucous gull	NA								
	pectoral sandpiper	NA								

TABLE 3-17. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF BIRDS AT THE WAINWRIGHT INSTALLATION (ORGANICS) (CONTINUED)

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Naphthalene	Lapland longspur	NA								
	brant	NA								
	glaucous gull	NA								
	pectoral sandpiper	NA								
Trimethylbenzene	Lapland longspur	NA								
	brant	NA								
	glaucous gull	NA								
	pectoral sandpiper	NA								

TABLE 3-18. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE WAINWRIGHT INSTALLATION (METALS)

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	TEST SPECIES	DOSE mg/kg-bw/day	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Aluminum	brown lemming	Acute systemic NOAEL; 100 day dietary study	rat	108	10	0.60	2	1	9.0	Domingo et al. 1987 in ATSDR 1990a
	arctic fox	Acute systemic NOAEL; 100 day dietary study	rat	108	10	2.70	2	1	2.0	Domingo et al. 1987 in ATSDR 1990a
	caribou	NOAEL; 20 day dietary study	sheep	48.6	1	1.17	2	1	21	Bailey 1977 in NAS 1980
Cadmium	brown lemming	Chronic NOAEL; two generation reproductive study	mouse	0.2	1	1.30	2	1	0.077	Schroeder and Mitchner 1971 in Opresko et al. 1994
	arctic fox	Chronic NOAEL; two generation reproductive study	mouse	0.2	1	5.82	2	1	0.017	Schroeder and Mitchner 1971 in Opresko et al. 1994
	caribou	Maximum tolerable dietary level (NOAEL)	sheep	0.02	1	1.17	2	1	0.0085	NAS 1980
Iron	brown lemming	Acute LD_{50}	rat	1,000	20	0.60	2	1	42	Shanas and Boyd 1969 in NAS 1980
	arctic fox	Acute LD_{50}	rat	1,000	20	2.70	2	1	9.3	Shanas and Boyd 1969 in NAS 1980
	caribou	NOAEL; maximum tolerable dietary level	sheep	20	1	1.17	2	1	8.5	NAS 1980
Lead	brown lemming	Chronic NOAEL, three generation reproductive study	rat	8	1	0.60	2	1	6.7	Azar et al 1973 in Opresko et al. 1994
	arctic fox	Chronic NOAEL, three generation reproductive study	rat	8	1	2.70	2	1	1.5	Azar et al. 1973 in Operesko et al. 1994

TABLE 3-18. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE WAINWRIGHT INSTALLATION (CONTINUED)

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	TEST SPECIES	DOSE mg/kg-bw/day	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
Lead (Continued)	caribou	Chronic NOAEL; 84 day dietary study	sheep	0.4	1	1.17	2	1	0.17	Fick et.al. 1976 in NAS 1980
Manganese	brown lemming	Chronic systemic NOAEL	rat	930	1	0.60	2	1	780	Hejtmancik et al. 1987 in ATSDR 1990b
	arctic fox	Chronic systemic NOAEL	rat	930	1	2.70	2	1	170	Hetjmancik et al. 1987 in ATSDR 1990b
	caribou	NOAEL; maximum tolerable dietary level	sheep	40	1	1.17	2	1	17	NAS 1980
Vanadium	brown lemming	Chronic reproductive NOAEL	rat	0.21	1	0.60	2	1	0.18	Domingo et al. in Opresko et al. 1994
	arctic fox	Chronic reproductive NOAEL	rat	0.21	1	2.70	2	1	0.039	Domingo et al. in Opresko et al. 1994
	caribou	NOAEL; maximum tolerable dietary level	cattle	2.0	1	0.58	2	1	1.7	NAS 1980
Zinc	brown lemming	Chronic reproductive NOAEL	rat	160	1	0.60	2	1	130	Schlicker and Cox 1968 in Opresko et al. 1994
	arctic fox	Chronic reproductive NOAEL	rat	160	1	2.70	2	1	30	Schlicker and Cox 1968 in Opresko et al. 1994
	caribou	Chronic NOAEL; 10 week physiological study	sheep	20	1	1.17	2	1	8.5	Ott et al. 1966 in NAS 1980

TABLE 3-19. TOXICITY REFERENCE VALUES FOR REPRESENTATIVE SPECIES OF MAMMALS AT THE WAINWRIGHT INSTALLATION (ORGANICS)

CHEMICAL OF CONCERN	REPRESENTATIVE SPECIES	STUDY TYPE	DOSE mg/kg-bw/day	TEST SPECIES	NOAEL UF	SCALING FACTOR	INTERSPECIES UF	PROTECTED SPECIES UF	TRV mg/kg-bw/day	REFERENCE
DRPH	brown lemming	LD_{50}	7,380	rat	20	0.60	2	1	310	Beck et al. 1982
	arctic fox	LD_{50}	7,380	rat	20	2.70	2	1	69	Beck et al. 1982
	caribou	LD_{50}	7,380	rat	20	7.24	2	1	26	Beck et al. 1982
Ethylbenzene	brown lemming	LD_{50}	5,460	rat	20	0.60	2	1	230	Clayton and Clayton 1981
	arctic fox	LD_{50}	5,460	rat	20	2.70	2	1	51	Clayton and Clayton 1981
	caribou	LD_{50}	5,460	rat	20	7.24	2	1	19	Clayton and Clayton 1981
Xylenes (total)	brown lemming	LD_{50}	4,300	rat	20	0.60	2	1	180	Clayton and Clayton 1981
	arctic fox	LD_{50}	4,300	rat	20	2.70	2	1	40	Clayton and Clayton 1981
	caribou	LD_{50}	4,300	rat	20	7.24	2	1	15	Clayton and Clayton 1981
Tetrachloroethene	brown lemming	NOAEL	1.4	mouse	1	1.30	2	1	0.53	Buben and O'Flaherty 1985 in Opresko et al. 1994
	arctic fox	NOAEL	1.4	mouse	1	5.82	2	1	0.12	Buben and O'Flaherty 1985 in Opresko et al. 1994
	caribou	NOAEL	1.4	mouse	1	15.59	2	1	0.045	Buben and O'Flaherty 1985 in Opresko et al. 1994
Naphthalene	brown lemming	NOAEL	50	rat	1	0.60	2	1	42	HSDB 1994
	arctic fox	NOAEL	50	rat	1	2.70	2	1	9.3	HSDB 1994
	caribou	NOAEL	50	rat	1	7.24	2	1	3.5	HSDB 1994
Trimethylbenzene	brown lemming	NA								
	arctic fox	NA								
	caribou	NA								

(1) The first step was to select an appropriate toxicity value from the scientific literature for each combination of chemical and representative or protected species. Test species most similar to the species of concern were preferred. A secondary emphasis was given to tests conducted over a significant portion of the animal's natural lifespan (e.g., chronic tests) when available.

(2) The second step was to modify the toxicity value, if necessary, through application of uncertainty factors associated with the quality of toxicity data to derive a NOAEL[4]. If a chronic NOAEL or NOEL was available, then the toxicity value was used with an uncertainty factor of one (i.e., no adjustment) because these values have the lowest uncertainty. If chronic data were unavailable, acute or subchronic toxicity data were modified by uncertainty factors to extrapolate to chronic effects. Based on Harding Lawson Associates (1992), the following strategy was derived for uncertainty factors for extrapolating study results to chronic NOAELs: 10 for chronic LOEL values, 10 for subchronic NOEL values, and 20 for subchronic LOEL values. LC_{50} and LD_{50} values are extrapolated to chronic NOAELs by a factor of 20.

(3) The third step is applicable only to terrestrial receptors. This step extrapolates the estimated NOAEL from the test species to a NOAEL for the species of concern using a body scaling factor. Klaassen et al. (1986) have indicated that dose expressed on a per unit surface area basis may be more appropriate than dose per unit body weight. The underlying assumption is that a toxicant acts on a physiologic surface and that the toxic effect increases as the ratio of chemical to surface area increases. The scaling factor (SIF) accounts for differences in the mass to surface area ratios between species. In this assessment the SIF was calculated using the following equation (Mantel and Schneiderman 1975):

$$\text{SIF} = (\text{weight of representative species/weight of test species})^{1/3}.$$

(4) An uncertainty factor of two was used to account for interspecies variation in sensitivity. This value is based on the methodology used in Harding Lawson Associates (1992).

(5) An uncertainty factor of two was used to account for additional sensitivity of state and/or federally protected species. This value is based on Harding Lawson Associates (1992). Migratory birds are federally protected and include all the representative avian and protected species selected for this assessment.

The methods of calculating the TRV for the terrestrial and aquatic receptors are as follows:

AQUATIC:

EFFECTIVE CONCENTRATION ÷ NOAEL UF ÷ INTERSPECIES UF = TRV

---

4 The highest concentration of a material in a toxicity test that has no statistically significant adverse effect on the exposed population of test organisms as compared with the next highest dose tested.

TERRESTRIAL:

(a) Convert test dose to a NOAEL:

DOSE ÷ **NOAEL UF** = Estimated NOAEL

(b) Adjust for body size difference between test species and ROC:

Estimated NOAEL ÷ **SCALING FACTOR**[5] = Scaled, estimated NOAEL

(c) Adjust for interspecific differences:

Scaled, estimated NOAEL ÷ **INTERSPECIES UF** = Species-specific, scaled, estimated NOAEL.

(d) Account for protected species status:

Species-specific, scaled, estimated NOAEL ÷ **PROTECTED SPECIES UF** = **TRV**

3.4 RISK CHARACTERIZATION FOR ECOLOGICAL RECEPTORS

In this section, potential risks to ecological receptors (representative species) are discussed. Potential risks to plants were evaluated based on the contaminant concentrations in the soil/sediment and information from the literature. Potential risks to aquatic organisms, birds, and mammals were estimated by comparing estimated exposures to TRVs (i.e., quotient method). The quotient method divides the estimated exposure concentration by the associated TRV to derive the HQ. If the HQ is less than 1.0, then adverse effects are not expected. Conversely, if the HQ is equal to or greater than 1.0, a potential for adverse effects exists. The confidence level of the risk estimate is increased as the magnitude of the HQ departs from 1.0. For example, there is greater confidence in an estimate where the HQ is 0.1 or 10, than in a HQ such as 0.9 or 1.1. The confidence level is also dependent on the uncertainties associated with the estimated exposure and the TRV for a given chemical-receptor combination.

The characterization of risk focuses on the assessment endpoints. These endpoints were selected and are discussed in keeping with the Framework for Ecological Risk Assessment guidance (EPA 1992b). The assessment endpoints for the Wainwright ERA are changes in:

- the populations of the plant representative species (*Carex* spp., *Salix* spp., *Eriophorum* spp. and *Vaccinium* spp.);
- the populations of aquatic representative species (*Daphnia* spp. and nine-spined stickleback);
- the populations of avian representative species (Lapland longspur, brant, glaucous gull and pectoral sandpiper); and

5 Scaling factors are presented in Appendix F

- the populations of mammalian representative species (brown lemming, arctic fox and barren-ground caribou).

Potential ecological risks are presented in the following sections: Section 3.4.1 addresses representative species of plants; Section 3.4.2 considers aquatic organisms; Section 3.4.3 addresses representative species of birds; and Section 3.4.4 discusses representative species of mammals. A discussion of potential future risks to ecological receptors is presented in Section 3.4.5. The HQs that represent potential risk estimates are summarized in Tables 3-21 and 3-22.

3.4.1 Potential Risks to Representative Species of Plants

In determining the risks to plants at the Wainwright installation, a qualitative comparison was made of soil and surface water contaminant concentrations and plant toxicity information in the literature. Table 3-20 summarizes these comparisons. There is a great deal of uncertainty in this phase of the assessment because of the differences in degree of uptake between plant species (Walker et al. 1978). However, the concentrations of contaminants onsite can be compared on the level of orders of magnitude. This comparison allows broad trends to be observed in order to determine whether a potential risk may exist.

Information is generally lacking concerning the toxicity of the COCs at Wainwright and how they relate to the representative species of plants. As a result, when comparisons of TRVs for site-specific species and chemicals are not possible, comparisons of related chemicals with other plant species are made. As seen in Table 3-20, the concentrations of metals, except aluminum and cadmium, found in the soils and water at Wainwright are at least one order of magnitude lower than reported toxicity values for various plants. Aluminum is elevated in water samples collected from the Landfill (LF05) (sample SW01 at 2,100 µg/L) and the Vehicle Storage Area (SS09) (sample SW01 at 9,700 µg/L). The average concentration of aluminum (2,000 µg/L) was not in excess of plant toxicity values; however, it was the same order of magnitude. As a result, a potential risk from aluminum may exist. However, because the aluminum was detected at elevated concentrations in just two locations, it is unlikely that these concentrations constitute a risk to plant populations at the Wainwright facility. The potential risk from cadmium is attributable to one sediment sample collected downstream from the Landfill. As a result, potential exposure of plants to cadmium is expected to be limited.

The comparisons presented in Table 3-20 are not definitive in judging the toxicity of metals to the specific representative plant species; noting the differences between exposure concentrations that pose risk and the concentration at the installations, but the risk to *Carex* spp., *Salix* spp., *Eriophorum* spp., and *Vaccinium* spp. is likely to be low. In addition, the concentrations of VOCs at the site are substantially lower than toxicity values reported by Galassi et al. (1988 in USACOE 1991) and Hutchinson et al. (1980 in USACOE 1991) and listed in Table 3-20. These VOCs are not expected to be present at significant levels in most plants because of their volatility, absorption to soil particles, metabolism, or degradation rates in soil (Kostecki and Calabrese 1989). Overall, the potential for adverse effects on representative species of plants at the six sites at the Wainwright installation can be characterized as low.

TABLE 3-20. COMPARISON OF CONCENTRATIONS OF POTENTIAL CONTAMINANTS TO TOXICITY INFORMATION FOR PLANTS AT THE WAINWRIGHT INSTALLATION

CHEMICAL (COC media)	PLANT	EXPOSURE LEVEL	EFFECT ON PLANT	WAINWRIGHT EXPOSURE	REFERENCE
ALUMINUM (COC in water)	Eurasian milfoil	2,500 µg/L in water	decreased root weights	2,000 µg/L	USACOE 1991
	rice/soybeans	8,000 µg/L in water	toxic shoot concentrations	2,000 µg/L	USACOE 1991
CADMIUM (COC in soil)	soybeans, wheat, lettuce	2.5 mg/kg	reduction in yield; discoloration	19 mg/kg	Haghiri 1973
IRON (COC in water and soil)	rice	100,000 - 500,000 µg/L >500,000 µg/L	toxic; highly toxic	28,000 µg/L	Foy et al. 1978
MANGANESE (COC in water and soil)	duckweed	31,000 µg/L in water	EC_{50}	690 µg/L	USACOE 1991
	terrestrial plants	1,500 - 3,000 mg/kg	phytotoxic	610 mg/kg	Kabata-Pendias and Pendias 1984
LEAD (COC in soil)	weed spp.	500 mg/kg in soil	reduced pollen germination	30 mg/kg	USACOE 1991
	weed spp.	46 mg/kg	normal germination	30 mg/kg	USACOE 1991
VANADIUM (COC in water)	diatom	100 µg/L	inhibition of growth	31 µg/L	Lee 1983
	algae	150 - 200 µg/kg	toxic threshold	31 µg/L	Lee 1983
ZINC (COC in water and soil)	several spp.	270 mg/kg (avg) in soil	phytotoxic	81 mg/kg	USACOE 1991
	Lemna minor	67,700 µg/L	EC50	670 µg/L	Brown and Rattigan 1979 in EPA 1987
VOCs	green algae	4,600 µg/L for ethylbenzene 2,290 µg/L for methylene chloride, in water	EC_{50}	821 µg/L as DRPH	USACOE 1991

3.4.2 Potential Risks to Representative Species of Aquatic Organisms

Estimates of exposure for aquatic organisms are based on the average concentrations of each COC in surface water samples (Section 3.1). The TRVs for aquatic species are presented in Table 3-21. The HQs were calculated by dividing the estimated exposure concentration by the TRV. Table 3-21 presents the results of the risk characterization for aquatic organisms. The following paragraphs summarize the potential risks to aquatic organisms from each COC in surface water (aluminum, iron, manganese, vanadium and zinc).

The HQs for aluminum in surface water were 20 and 22 for *Daphnia* spp. and stickleback, respectively. These HQs indicate that a potential risk exists for aquatic organisms from aluminum concentrations in surface water. Aluminum was detected in three surface water samples. In particular, sample SW01 from the Vehicle Storage Area was elevated (9,700 μg/L). Sample SW01 from the Landfill was also high at 2,100 μg/L. The third sample, SW02 from the Landfill, was much lower, at 180 μg/L. No metals were detected in the remainder of the samples. It is important to note, however, that the detection limit for aluminum in surface water (100 μg/L) is above the action level. As a result, the non-detected samples may contribute slightly to the potential hazard from aluminum. This may be mitigated, however, by the fact that "total" metal concentrations were used to estimate the HQ. Total metals are higher than dissolved metals (the bioavailable fraction) and this is likely to overestimate the HQ. The site-specific risks at the Wainwright facility are likely to be lower than presented in these risk estimates because of the nature of the drainage areas at the installation. The surface water at the Vehicle Storage Area is not a significant route of exposure for the nine-spined stickleback as they are shallow ephemeral ponds that freeze to the bottom in the winter. As a result, *Daphnia* spp. is the only representative species likely to be exposed in the Vehicle Storage Area. Conditions at the Landfill are similar. Discharge from the Landfill joins shallow intermittent streams which are not likely to support the nine-spined stickleback. As a result, the risk to aquatic organisms from aluminum is very limited.

The iron HQs are 28 for both *Daphnia* spp. and the nine-spined stickleback. Concentrations of iron were in excess of the TRV in every sample. The most elevated concentrations were at the Vehicle Storage Area (130,000 μg/L in SW01) and the Landfill (23,000 μg/L in SW01). Although these elevated HQs may indicate risk, the nine-spined stickleback is not likely to be found in the locations where iron concentrations are elevated (i.e., intermittent, seasonal drainages). The HQs are based on total iron concentrations; as a result, the dissolved fraction may be somewhat lower. Therefore, the risk from iron to aquatic organisms is also limited.

Manganese HQs range from 1.7 for Daphnia spp. to 9.5 for the nine-spined stickleback. This indicates a potential risk to these species; however, some mitigating factors should be considered. The average manganese concentration at the Wainwright installation of 690 μg/L is only slightly greater than the maximum background concentration of 510 μg/L. In addition, these background concentrations approximate normal manganese concentrations in surface waters (USGS 1985). Therefore, it is likely the potential risk to aquatic species from manganese at the Wainwright installation is minimal.

TABLE 3-21. RISK CHARACTERIZATION OF REPRESENTATIVE SPECIES OF AQUATIC ORGANISMS AT THE WAINWRIGHT INSTALLATION

SPECIES	ESTIMATED EXPOSURE CONCENTRATION μg/liter	TRV μg/liter	HAZARD QUOTIENT
ALUMINUM			
nine-spined stickleback	2,000	92	22
Daphnia spp.	2,000	102	20
IRON			
nine-spined stickleback	28,000	1,000	28
Daphnia spp.	28,000	1,000	28
MANGANESE			
nine-spined stickleback	690	73	9.5
Daphnia spp.	690	410	1.7
VANADIUM			
nine-spined stickleback	31	160	0.2
Daphnia spp.	31	76	0.4
ZINC			
nine-spined stickleback	670	70	9.6
Daphnia spp.	670	47	14

The toxicity of zinc is dependent upon water hardness. Generally, as hardness increases, toxicity of zinc decreases; however, site-specific hardness was not reported for Wainwright installation. The zinc TRV for *Daphnia* spp. used in this assessment was based on a water hardness of 104 mg/L as $CaCO_3$ (the hardness for the stickleback TRV was not reported). Zinc HQs ranged from 9.6 for the nine-spined stickleback to 14 for *Daphnia* spp., which indicates a potential risk to these species. Zinc was detected at elevated concentrations at the Vehicle Storage Area, Landfill, and Garage. All of the samples were collected from shallow ponds that are not likely to be used by fish species. As a result, the overall risks to on aquatic species may be generally classified as low.

3.4.3 Potential Risks to Representative Species of Birds

The HQs for all COCs except cadmium and iron are below 1.0 for all species of birds evaluated, (see Table 3-22). Cadmium presented a slight potential risk to the brant (HQ of 1) and pectoral sandpiper (HQ of 2). The potential risk from cadmium is due to one detection at the Landfill in sediment sample SD02 at a concentration of 72 mg/kg. The potential for adverse effects on these species is attributable to the large fraction of sediment they are expected to ingest while foraging. Iron presented a potential risk to Lapland longspur (HQ of 3), brant (HQ of 4), and pectoral sandpiper (HQ of 40). Iron was elevated above background concentrations in both surface water (average concentration of 28,000 μg/L) and soil (57,000 mg/kg). The highest iron concentrations in soil were located in the Drum Storage Area (sample S04 at 110,000 mg/kg), the Landfill (sample S04-1.5 at 99,000 mg/kg), and the Garage (sample SD01 at 114,000 mg/kg). Of these samples, only the one from the Landfill is not likely to be actually within the potential exposure pathway of representative species of birds as the sample was collected at a depth of 1.5 feet. Based on the HQs presented above, the pectoral sandpiper is at moderate risk from iron and cadmium exposure. However, the potential hazard posed by cadmium is mitigated by the "hot spot" nature of the contamination. It is unlikely that sandpipers will be exposed in the vicinity of the Landfill discharge in any frequency or duration that could pose a substantial risk to the population. The risks to other birds may be qualified as low. However, it is important to note that a portion of the birds' diets (invertebrates) was not quantified in this assessment, which may result in an underestimate of potential exposure to metals. .

No toxicity values were available for several COCs detected in soils: ethylbenzene, tetrachloroethene, naphthalene, and trimethylbenzene. The sampling detected ethylbenzene in 4 of 39 samples, tetrachloroethene in 4 of 29 samples, naphthalene in 3 of 11 samples, and trimethylbenzene in 4 of 11 soil/sediment samples. The frequency of detections, combined with the relatively low concentrations and resulting minimal exposures (see Table 3-22, estimated daily dose column) indicate that the risk from these chemicals is likely low.

3.4.4 Potential Risks to Representative Species of Mammals

HQs for the arctic fox and the caribou were less than one for all COCs. The only risk to representative species of mammals at the Wainwright installation is to the brown lemming, which have a potential risk from cadmium and iron (HQs of 60 and 20, respectively). It is important to note that cadmium was detected in one sediment sample downstream from the Landfill. All other soil/sediment samples were non-detects for cadmium. This cadmium "hot spot" is within a

TABLE 3-22. RISK CHARACTERIZATION OF REPRESENTATIVE BIRDS AND MAMMALS AT THE WAINWRIGHT INSTALLATION

SPECIES	ESTIMATED DAILY DOSE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
ALUMINUM			
Lapland longspur	2×10^{-1}	50	4×10^{-3}
brant	3×10^{-2}	14	2×10^{-3}
glaucous gull	4×10^{-3}	13	3×10^{-4}
pectoral sandpiper	3×10^{-1}	35	7×10^{-3}
brown lemming	1×10^{-1}	9.0	1×10^{-2}
arctic fox	2×10^{-3}	2.0	9×10^{-4}
caribou	1×10^{-3}	21	6×10^{-5}
CADMIUM			
Lapland longspur	4×10^{-1}	0.41	9×10^{-1}
brant	1×10^{-1}	0.11	**1×10^{0}**
glaucous gull	5×10^{-3}	0.11	5×10^{-2}
pectoral sandpiper	6×10^{-1}	0.29	**2×10^{0}**
brown lemming	5×10^{0}	0.077	**6×10^{1}**
arctic fox	3×10^{-4}	0.017	1×10^{-2}
caribou	3×10^{-3}	0.0085	3×10^{-1}
IRON			
Lapland longspur	2×10^{2}	55	**3×10^{0}**
brant	7×10^{1}	15	**4×10^{0}**
glaucous gull	9×10^{0}	14	6×10^{-1}
pectoral sandpiper	1×10^{3}	38	**4×10^{1}**
brown lemming	7×10^{2}	42	**2×10^{1}**
arctic fox	9×10^{-1}	9.3	9×10^{-2}
caribou	4×10^{-1}	8.5	5×10^{-2}
LEAD			
Lapland longspur	1×10^{-1}	14	8×10^{-3}
brant	5×10^{-2}	3.7	1×10^{-2}

NC Not calculated because toxicity values were not available.

TABLE 3-22. RISK CHARACTERIZATION OF REPRESENTATIVE BIRDS AND MAMMALS AT THE WAINWRIGHT INSTALLATION (CONTINUED)

SPECIES	ESTIMATED DAILY DOSE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
glaucous gull	5×10^{-3}	3.5	1×10^{-3}
pectoral sandpiper	8×10^{-1}	9.5	8×10^{-2}
brown lemming	9×10^{-1}	6.7	1×10^{-1}
arctic fox	4×10^{-4}	1.5	3×10^{-4}
caribou	5×10^{4}	0.17	3×10^{-3}
MANGANESE			
Lapland longspur	6×10^{0}	140	5×10^{-2}
brant	3×10^{0}	37	7×10^{-2}
glaucous gull	1×10^{-1}	36	4×10^{-3}
pectoral sandpiper	2×10^{1}	95	2×10^{-1}
brown lemming	7×10^{1}	780	9×10^{-2}
arctic fox	9×10^{-3}	170	6×10^{-5}
caribou	4×10^{-2}	17	2×10^{-3}
VANADIUM			
Lapland longspur	3×10^{-3}	1.4	2×10^{-3}
brant	4×10^{-4}	0.38	1×10^{-3}
glaucous gull	7×10^{-5}	0.37	2×10^{-4}
pectoral sandpiper	4×10^{-3}	0.98	4×10^{-3}
brown lemming	2×10^{-3}	0.18	1×10^{-2}
arctic fox	3×10^{-5}	0.039	7×10^{-4}
caribou	2×10^{-5}	1.7	1×10^{-5}
ZINC			
Lapland longspur	4×10^{0}	14	3×10^{-1}
brant	2×10^{0}	3.7	4×10^{-1}
glaucous gull	4×10^{-2}	3.6	1×10^{-2}
pectoral sandpiper	4×10^{0}	9.5	4×10^{-1}
brown lemming	5×10^{1}	130	4×10^{-1}

NC Not calculated because toxicity values were not available.

TABLE 3-22. RISK CHARACTERIZATION OF REPRESENTATIVE BIRDS AND MAMMALS AT THE WAINWRIGHT INSTALLATION (CONTINUED)

SPECIES	ESTIMATED DAILY DOSE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
arctic fox	2×10^{-3}	30	6×10^{-5}
caribou	3×10^{-2}	8.5	4×10^{-3}
DRPH			
Lapland longspur	1×10^{1}	520	2×10^{-2}
brant	5×10^{0}	140	3×10^{-2}
glaucous gull	5×10^{-1}	140	4×10^{-3}
pectoral sandpiper	8×10^{1}	360	2×10^{-1}
brown lemming	8×10^{1}	310	3×10^{-1}
arctic fox	5×10^{-2}	69	7×10^{-4}
caribou	4×10^{-2}	26	2×10^{-3}
ETHYLBENZENE			
Lapland longspur	7×10^{-3}	NC	NC
brant	3×10^{-3}	NC	NC
glaucous gull	9×10^{-5}	NC	NC
pectoral sandpiper	1×10^{-2}	NC	NC
brown lemming	8×10^{-2}	230	4×10^{-4}
arctic fox	5×10^{-6}	51	9×10^{-8}
caribou	5×10^{-5}	19	3×10^{-4}
NAPHTHALENE			
Lapland longspur	2×10^{-3}	NC	NC
brant	6×10^{-4}	NC	NC
glaucous gull	3×10^{-5}	NC	NC
pectoral sandpiper	3×10^{-3}	NC	NC
brown lemming	2×10^{-2}	42	5×10^{-4}
arctic fox	1×10^{-6}	9.3	2×10^{-7}
caribou	1×10^{-5}	3.5	4×10^{-6}

NC Not calculated because toxicity values were not available.

TABLE 3-22. RISK CHARACTERIZATION OF REPRESENTATIVE BIRDS AND MAMMALS AT THE WAINWRIGHT INSTALLATION (CONTINUED)

SPECIES	ESTIMATED DAILY DOSE mg/kg-bw/day	TRV mg/kg-bw/day	HAZARD QUOTIENT
TETRACHLOROETHENE			
Lapland longspur	5×10^{-2}	NC	NC
brant	2×10^{-2}	NC	NC
glaucous gull	5×10^{-4}	NC	NC
pectoral sandpiper	5×10^{-2}	NC	NC
brown lemming	6×10^{-1}	0.53	**1×10^{0}**
arctic fox	2×10^{-5}	0.12	2×10^{-4}
caribou	4×10^{-4}	0.045	8×10^{-3}
TRIMETHYLBENZENE			
Lapland longspur	1×10^{-2}	NC	NC
brant	5×10^{-3}	NC	NC
glaucous gull	3×10^{-4}	NC	NC
pectoral sandpiper	4×10^{-2}	NC	NC
brown lemming	2×10^{-1}	NC	NC
arctic fox	2×10^{-5}	NC	NC
caribou	1×10^{-4}	NC	NC
XYLENES			
Lapland longspur	3×10^{-2}	26	1×10^{-3}
brant	1×10^{-2}	7.2	2×10^{-3}
glaucous gull	4×10^{-4}	6.8	5×10^{-5}
pectoral sandpiper	4×10^{-2}	18	2×10^{-3}
brown lemming	4×10^{-1}	180	2×10^{-3}
arctic fox	1×10^{-5}	40	4×10^{-7}
caribou	3×10^{-4}	15	2×10^{-5}

NC Not calculated because toxicity values were not available.

surface water drainage area, so it is unlikely that a brown lemming would be significantly exposed to this source. In addition, there is a significant amount of uncertainty in the plant bioconcentration coefficient (B_v) used to estimate cadmium uptake by the plants in the lemming diet. These circumstances suggest that the potential for adverse effects from the exposure of lemmings to cadmium may be negligible.

Iron also presented a slight risk to lemmings based on the HQ of 20. Iron is an essential nutrient that is regulated by mammals. As a result, although exposure concentrations at the Wainwright facility are elevated, it is unknown how much iron is actually necessary in the lemming's diet, and these levels may not produce a toxic response. In addition, there is some uncertainty surrounding the uptake of iron by plants and the subsequent bioavailability of the iron to the herbivore. Studies have shown that inorganic iron is more available than iron present in either the grasses or legumes (NAS 1980). The assumptions used in this risk assessment may overestimate the potential risks from iron.

Because the necessary toxicity values were not available for mammals in the published literature, HQs were not calculated for 1,2,4- and 1,3,5-trimethylbenzene. These chemicals are not expected to contribute significantly to the potential for adverse effects in mammals because they were observed at relatively low concentrations and detection frequencies. Furthermore, the toxicity of these compounds for ecological receptors is expected to be low (Verschueren 1983).

3.4.5 Potential Future Risks

Since this ERA assumed that all areas of the Wainwright installation are suitable for use by representative species, the potential future risk should not increase. In general, there are no chemicals that are likely to bioconcentrate, and COC concentrations should decline over time. As a result, the potential risks are likely to decline over time, and future risks associated with Wainwright are considered low.

3.5 ECOLOGICAL RISK ASSESSMENT UNCERTAINTY ANALYSIS

As with any risk assessment, there is great uncertainty associated with the estimates of ecological risk for the sites at the Wainwright installation. The risk estimates are based on a number of assumptions regarding exposure and toxicity. In general, the primary sources of uncertainty are the following:

- Environmental Sampling and Analysis;
- Selection of COCs;
- Selection of Representative Species;
- Exposure Parameter Estimation; and
- Toxicological Data.

A complete understanding of the uncertainties associated with risk estimates is critical to placing the predicted risks in proper perspective. The most significant sources of uncertainty associated

with the risk estimates for the Wainwright installation sites are summarized in the following sections.

3.5.1 Environmental Sampling and Analysis

The principal source of uncertainty in the analytical data (for the ERA) stems from the sampling approach and the subsequent calculation of exposure concentrations. Sampling at the Wainwright installation was conducted in a systematic manner, designed to characterize localized contaminated areas or "hot spots"[6]. The sites' potential source areas are therefore well characterized; however, there are limited data regarding the peripheral areas (areas to which ecological receptors are most likely to be exposed). In order to compensate for this non-random sampling methodology in the calculation of exposure concentrations, the exposure assessment used the average concentration of COCs across the site.

The methods of calculating the average concentrations were the same for organic and inorganic data; non-detect chemicals were entered at one-half of the quantitation limit, as per EPA guidance (EPA 1989). Because sampling was designed to characterize "hot spots" at each of the sites, it was generally concentrated in areas of the site where significant contamination exists or was suspected. Therefore, the average concentrations of COCs tend to be biased high. The use of total metal concentrations in surface water is a conservative approach because dissolved metal concentrations are generally significantly lower.

An additional factor related to the analysis of surface water samples and the associated risk estimates should be considered. The detection limits of several metals were higher than the action levels used to screen the chemicals. For example, the detection limit of aluminum is <100 µg/L, and the action level is 87 µg/L. Therefore, non-detected concentrations of aluminum and other metals, including cadmium, chromium, copper, lead, selenium, and silver, may be sufficient to elicit adverse effects in aquatic organisms. This probably contributes a low level of uncertainty to the overall risk estimate because surface water pathways at the Wainwright installation are unlikely to be significant routes of exposure to representative species other than *Daphnia* spp.

Further, there is uncertainty inherent in using measurements of DRPH, GRPH, and RRPH for risk assessments. The analytical techniques are not specific to petroleum (i.e., they detect other organics, including naturally-occurring ones) (Von Burg 1993). Moreover, the toxicity of these groups of petroleum hydrocarbons is determined by the toxicity of their individual constituents. When petroleum compounds are released to the environment, they tend to weather or transform readily. For example, the lighter fractions (such as BTEX) will volatilize to the atmosphere more readily than the heavier fractions (such as decane, pyrene, or benzo(a)pyrene). The lighter fractions are thought to be the more toxic (Wong et al. 1981; O'Brien 1978; Kauss and Hutchinson 1975; and Soto et al. 1975). Therefore, the toxicities of DRPH, GRPH, and RRPH are expected to change over time depending upon the attenuation mechanisms occurring in the environment. As a result, the toxicity of the petroleum hydrocarbons detected at the Wainwright

6 For example, cadmium in soils/sediment was detected at one location (sample SD02 from the Landfill).

installation is unknown. Use of toxicity values reported in the literature probably contributes to an overestimation of the risk because it is likely that the most toxic components of the mixtures have volatilized to the atmosphere over time.

3.5.2 Selection of Chemicals for Evaluation

The selection of COCs in the ERA was based upon a comparison to background concentrations and action levels, and an evaluation of the frequency of detection. For certain chemicals, no action levels were available and action levels for related compounds were used. This introduces uncertainty into the risk assessment as actual toxicity may be different from that of the surrogate chemical. Overall, however, the process provided a conservative screen of COCs, and it is unlikely that any chemicals presenting an ecological risk were omitted.

3.5.3 Selection of Representative Species

The selection of representative species in the ERA also introduces uncertainty into the risk estimates. No site-specific biological survey was conducted at the Wainwright facility, with the exception of a survey for spectacled and Steller's eiders (Alaska Biological Research 1994). As a result, it is not known whether the representative species actually occur at the site. However, the uncertainty introduced into the risk estimate by this route is likely to be low. The purpose of ERAs is not to survey the biota at a site, but to estimate the risks to species that may inhabit the area. Surrogate species are commonly used, and even if the representative species do not dwell at the Wainwright facility, the estimates in this report will provide a sound measure of the potential risks to species inhabit the area.

3.5.4 Exposure Assessment

Exposures were estimated from literature-based life history information for the representative species. There is moderate uncertainty associated with the exposure information. Food and water ingestion rates were not available for some animals and were estimated from regression equations. Incidental ingestion of soils and sediments may occur while animals are foraging in these media, but the amount is uncertain. In addition, samples were collected around buildings and other structures that provide habitat of limited quality; this tends to overestimate exposure. Further, there are significant uncertainties associated with the estimates of how extensively a receptor will use a site, which were based on home range information. As noted in the discussion of Estimation of Percent Ingested Onsite, Section 3.2.7.2, the conversion of population density values to home ranges adds further uncertainty but was necessary because home range data are lacking for some of the representative species.

There is some uncertainty associated with the diet compositions estimated from published information. As the numbers of prey increase, predator populations may experience numerical and density increases well beyond the values reported in the literature [e.g., unpredictable fluctuations in the populations of the brown lemmings and their predators (i.e., arctic fox, glaucous gull)]. When prey populations decrease, predation pressure can shift to diet items that are not considered "normal", and do not represent dietary intakes reported in the literature. Wildlife and its interactions with the environment are dynamic. Stochastic events, natural or

anthropogenic, may cause behavior and/or habits to differ markedly from the "expected or norm". Deviations from typical behavior cause uncertainty in the evaluation of wildlife and ecosystems.

There is also uncertainty associated with exposure estimates for plants. Plant uptake of a COC was derived from a regression equation using the K_{ow} of the COC (Table 3-6) to estimate the concentration of chemicals in the vegetative portion of plants. Actual concentrations of the COC in plant tissue will vary depending upon actual chemical uptake, species of plant, and other site-specific factors (such as soil organic carbon). It is important to note that maximum acceptable tissue concentrations in plants were not available for comparison with these estimated concentrations. As a result, it is uncertain whether the estimates are phytotoxic. However, the overall effect of this source of uncertainty in the risk assessment is low, as is the ecological risk to plants.

In addition, the only component in the diet of representative species evaluated quantitatively was the ingestion of plants. Ingestion of animal prey (e.g., the diet of the arctic fox) was not quantified. This may slightly underestimate risk for species that rely on animal items in their diet.

3.5.5 Toxicological Data

One of the largest sources of uncertainty in risk assessment is from the toxicological data. Often there are no relevant studies for the representative species or endpoints, and extrapolations introduce uncertainty into the risk estimate. Uncertainty factors are incorporated into the calculation of TRVs to include a margin of error in the risk estimate and determine at a "safe" level of exposure to which onsite exposure concentrations may be compared. These techniques introduce into the risk assessment a tendency to overestimate the risk.

For some chemicals, no toxicity information was available (ethylbenzene, tetrachloroethene, and naphthalene for birds; and trimethylbenzene for birds and mammals). As a result, these compounds were not evaluated quantitatively in the risk assessment, and the risks may be underestimated. However, the low concentrations and low frequency of detection of these compounds (as discussed in Section 3.1) indicate that the uncertainty associated with this factor is low.

Toxicity values for plants, water, soils, and sediments are based on the literature. In soils and sediments, toxicity is affected by the bioavailability of a given chemical, and toxicity of metals in water is based, in part, upon the speciation of the element. As a result, site-specific bioavailability and toxicity may differ from those in published studies. In addition, the sensitivity of receptors on site may be different from the sensitivity of the species reported in the literature. This contributes to the overall uncertainty of the risk assessment.

There is also a great deal of uncertainty in assessing the toxicity of a mixture of chemicals. In this ERA, the effects of exposure from each contaminant have been considered separately. However, these substances occur together at the site, and organisms may be exposed to mixtures of the chemicals. Prediction of how these mixtures of toxicants will interact must be based on an understanding of the mechanisms of such interactions. Interactions of the individual components of chemical mixtures may occur during absorption, distribution, metabolism,

excretion, or activity at the receptor site. Individual compounds may interact chemically, yielding a new toxic component or causing a change in the biological availability of an existing component, or may interact by causing different effects at different receptor sites. Suitable data are not currently available to characterize the effects of chemical mixtures rigorously, so chemicals present at the site were evaluated independently. This approach of assessing risk associated with mixtures of chemicals does not account for any additive, synergistic, or antagonistic interactions among the chemicals considered. However, as discussed in Section 3.6, the risk assessment yielded a low potential for ecological risks, and it is unlikely that any additive effects of chemicals are a concern.

3.6 SUMMARY OF ECOLOGICAL RISK

The potential risks to ecological receptors are summarized in this section based on the information presented in Sections 3.1 through 3.4. Conclusions regarding potential risks must be viewed in the context of uncertainties associated with the assessment (Section 3.5) and the available risk information. The available risk information includes chemical data, exposure estimates, and literature-based toxicity information.

Table 3-23 presents a summary of the ecological risks at the Wainwright installation. The table includes the potential risk to each ecological group evaluated, the COC that contributed to the risk, and the site(s) where the COCs were detected at relatively high concentrations.

TABLE 3-23. SUMMARY OF POTENTIAL ECOLOGICAL RISKS

ECOLOGICAL GROUP	POTENTIAL RISK	COC	SITES
Plants	Low	Cadmium in soil/sediments	Landfill (LF05)
Aquatic Organisms	Low	Aluminum, iron, manganese and zinc in surface water	Landfill (LF05) Vehicle Storage Area (SS09) Garage (SS07)
Birds	Low	Cadmium in soil/sediment; iron in soil/sediment	Landfill (LF05) Vehicle Storage Area (SS09) Garage (SS07) Drum Storage Area (ST02)
Mammals	Low - arctic fox, caribou Moderate - brown lemming	Lead in soil/sediment	Landfill (LF05) Vehicle Storage Area (SS09) Garage (SS07) Drum Storage Area (ST02)

3.6.1 Potential Risks to Representative Plants

A qualitative comparison was conducted of onsite soil and surface water concentrations with plant toxicity information. The risk to plants is characterized by using comparative information from the literature and BCF (B_v). Based on the qualitative comparison, the risks to plants (with the exception of cadmium concentrations in sediment) is low. It is important to consider that cadmium was detected in only one sediment sample associated with the Landfill that is considered indicative of a "hot spot" of contamination and not representative of the Wainwright facility.

3.6.2 Potential Risks to Representative Aquatic Species

Potential risks to aquatic species were evaluated by comparing toxicity information from the literature with the average exposure concentrations of potential contaminants in surface water. HQs for aquatic organisms indicate that risks may exist from aluminum, iron, manganese, and zinc. The HQs for fish and aquatic invertebrates for aluminum are 22 and 20, for manganese are 9.5 and 1.7, and for zinc are 9.6 and 14, respectively. The HQ for iron for aquatic organisms is 28. Concentrations of metals were elevated above background concentrations and above action levels. The risk estimates represent a moderate potential for adverse effects to aquatic receptors. It is important to qualify this risk in terms of site-specific conditions. The elevated concentrations were in samples collected from seasonal drainages that freeze solid in the winter, and are not likely to be suitable habitat for fish, but may provide habitat for invertebrates. In addition, the potential risks to aquatic species was based on total metal concentrations; use of dissolved metal concentrations would probably result in lower potential risk estimates. Considering site-specific factors, the overall risk to aquatic organisms at the Wainwright facility is considered low.

3.6.3 Potential Risks to Representative Species of Birds and Mammals

The risks to representative species of birds and mammals were evaluated using the quotient method, which compares the estimated dose with the TRV. The resulting HQs indicate that the overall risks to birds and mammals are low. Iron presented a low to moderate potential risk for birds (HQ ratio ranging from 3 for the Lapland longspur to 40 for the pectoral sandpiper), and cadmium also contributed to the risk for birds (HQs of one for the brant to two for the pectoral sandpiper). The risk from cadmium is attributable to a sediment sample collected downstream from the Landfill (LF05). No samples were collected upstream or downstream of this sample location, so it is difficult to speculate on the extent of cadmium contamination, but it seems reasonable that it is restricted to the vicinity of the Landfill. Given the home range requirements of the representative species, it is unlikely that any bird would be receiving all of its exposure to cadmium from that one location, and the risk to birds from cadmium is considered low.

HQs for mammalian representative species were all below one, with the exception of cadmium and iron exposure for the brown lemming. Due to the "hot spot" nature of cadmium concentrations discussed above, and the fact that the cadmium was detected in a sediment sample, it is unlikely that cadmium presents an actual threat to the lemming. Iron was elevated above background concentrations in both surface water (average concentration of 28,000 µg/L) and soil (average concentration 57,000 mg/kg). Iron is a nutrient that is regulated by mammals,

so although exposure concentrations at the Wainwright facility are elevated, these levels may not produce a toxic response. In addition, there is some uncertainty surrounding the uptake of iron by plants and the subsequent bioavailability of the iron to the herbivore. Studies have shown that inorganic iron is more available than iron present in either the grasses or legumes (NAS 1980) and the assumptions in this risk assessment may overestimate potential risk to the brown lemming from iron.

The objective of this ERA was to evaluate the potential risk to the representative species at the Wainwright DEW Line installation. This assessment indicates that, although there are a few instances of minimal potential risk to individual species, overall the potential risks presented by the COCs are very low.

THIS PAGE INTENTIONALLY LEFT BLANK

4.0 REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. Toxicological Profile for Zinc. Draft Report. U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 1990a. Toxicological Profile for Aluminum. Draft Report. U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 1990b. Toxicological Profile for Manganese. Draft Report. U.S. Department of Health and Human Services.

Agency for Toxic Substances and Disease Registry (ATSDR). 1991. Toxicological Profile for Vanadium. Draft Report. U.S. Department of Health and Human Services.

Alaska Biological Research. 1994. Spectacled and Steller's Eiders Surveys at 11CEOS Remote Sites in Alaska, 1994. Fairbanks, Alaska. 30 September 1994.

Alaska Department of Environmental Conservation. 1991. Interim Guidance for Non-UST Contaminated Soil Cleanup Levels. Guidance No. 001 - Revision No. 1, 17 July 1991. Table 2 (North Slope Numeric Cleanup Levels).

Albers, P.H. 1977. Effects of External Applications of Fuel Oil on Hatchability of Mallard Eggs. Pages 158-163 in D.A. Wolfe (Ed). Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems And Organisms. Pergamon Press, New York.

Allaway, W.H. 1968. Agronomic Controls Over the Environmental Cycling of Trace Elements. In: A.G. Norman (Ed.). Advances in Agronomy. Academic Press, New York, New York. 20:235-274.

Ambrose, S. 1994. Personal communication with S. Ambrose, a threatened and endangered species specialist with the U.S. Fish and Wildlife Service. Don Kellett, ICF Kaiser Engineers, Lakewood, Colorado. 31 March 1994.

American Petroleum Institute (API). 1994. Results of Toxicological Studies Conducted for the American Petroleum Institute. API Publication No. 45591. January 1994.

Aquatic Information Retrieval (AQUIRE). 1990. Computerized Database. Chemical Information System, Inc., Baltimore, Maryland.

Aquatic Information Retrieval (AQUIRE). 1994. Computerized Database. Chemical Information System, Inc., Baltimore, Maryland.

Armstrong Laboratory. 1994. Evaluation of the Total Petroleum Hydrocarbon Standard at Jet Fuel Contaminated Air Force Sites. Prepared by EA Engineering, Science and Technology Inc. for Armstrong Laboratory, Brook AFB, Texas. January.

Baes, C.F., III, R.D. Sharp, A.L. Sjoreen, and R.W. Shor. 1984. A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture. Oak Ridge National Laboratory. Prepared for the U.S. Department of Energy, Contract No. DE-AC05-84OR21400.

Beck, L.S., D.I. Hepler, and K.L. Hansen. 1982. The Acute Toxicology of Selected Petroleum Hydrocarbons. Pages 1-12 in H.N. MacFarland, C.E. Holdsworth, J.A. MacGregor, R.W. Call, and M.L. Kaen (Eds). Proceedings of the Symposium - The Toxicology of Petroleum Hydrocarbons. American Petroleum Institute. Washington, D.C.

Belopol'skii, L.O. 1961. Ecology of Sea Colony Birds of the Barents Sea. Translated from the Russian original. Israel Program for Scientific Translations. Jerusalem, Israel.

Bergman, R.D., R.L. Howard, K.F. Abraham, and M. W. Weller. 1977. Water Birds and Their Wetland Resources in Relation to Oil Development at Storkersen Point, Alaska. Resource Publication 129. U.S. Fish and Wildlife Service. Washington D.C.

Beyer, N., E. Conner, and S. Gerould. 1994. Estimates of Soil Ingestion by Wildlife. J. Wildl. Manage. 58(2):375-382.

Bott, T.L. and K. Rogenmuser. 1978. Effects of No. 2 Fuel Oil, Nigerian Crude Oil, and Used Crankcase Oil on Attached Algal Communities: Acute and Chronic Toxicity of Water-Soluble Constituents. Applied and Environmental Microbiology. November 1978:673-682.

Brewster, K. 1995. Personal communication with North Slope Borough oral historian.

Budavari, S. (Ed). 1989. The Merck Index - Encyclopedia of Chemicals, Drugs and Biologicals. Rahway, New Jersey: Merck and Co., Inc., p. 595.

Burt, W.H. and R.P. Grossenheider (Eds). 1976. A Field Guide to the Mammals. Third Edition. Houghton-Mifflin Co., Boston, Massachusetts.

Calder, W.A. and E.J. Braun. 1983. Scaling of Osmotic Regulation in Mammals and Birds. Am J. Physiol. 244:R601-R606.

Cameron, R.D., D.J. Reed, J.R. Dau, and W.T. Smith. 1992. Redistribution of Calving Caribou in Response to Oil Field Development on the Arctic Slope of Alaska. Arctic 45(4):338-342.

Chance, N. 1990. The Inupiat and Arctic Alaska: An Ethnography of Development. Holt, Rinehart, and Winston, New York. Pp. 241.

Chappell, M.A. 1980. Thermal Energetics and Thermoregulatory Costs of Small Arctic Mammals. J. Mamm. 61(2):278-291.

Chesemore, D.L. 1967. Ecology of the Arctic Fox in Northern and Western Alaska. M.S. Thesis, University of Alaska, Fairbanks.

Clayton, G.D. and F.E. Clayton (Eds). 1981-1982. Patty's Industrial Hygiene and Toxicology: Volume 2A, 2B, 2C: Toxicology. Third Edition. New York: John Wiley Sons, p. 3292.

Cuccarese, S.V., M.F. Arend, R.J. Hensel, and P.O. McMillan. 1984. Biological and Socioeconomic Systems of the BAR-M, POW-1, LIZ-3A and SI-1 North Warning System Sites, Alaska. AEIDC. University of Alaska, Anchorage.

Custance, S.R., P.A. McCaw, A.C. Kopf, and M.J. Sullivan. 1992. Environmental Fate of the Chemical Mixtures: Crude Oil, JP-5, Mineral Spirits and Diesel Fuel. Journal of Soil Contamination 1(4):379-386.

Custer, T.W. and F.A. Pitelka. 1977. Demographic Features of a Lapland Longspur Population Near Barrow, Alaska. Auk 94:505-525.

Custer, T.W. and F.A. Pitelka. 1978. Seasonal Trends in Summer Diet of the Lapland Longspur Near Barrow, Alaska. Condor 80:295-301.

Delorme Mapping. 1992. Alaska Atlas and Gazetteer. First Edition. Second Printing.

Delos, C. 1995. Personal communication with Charles Delos, United States Environmental Protection Agency. Karen Prochnow, ICF Kaiser Engineers. 25 April 1995.

Derksen, D.V., T.C. Rothe, and W.D. Eldridge. 1981. Use of Wetland Habitats by Birds in the National Petroleum Reserve - Alaska. U.S. Fish and Wildlife Service, Resource Publication 141. Washington, D.C.

DiGuilio, R.T. and Scanlon, R.F. 1984. Sublethal Effects of Cadmium Ingestion on Mallard Ducks. Arch. Environ. Contam. Toxicol. 13:765.

Doudoroff, P. and M. Katz. 1953. Critical Review of Literature on the Toxicity of Industrial Wastes and their Components to Fish. II. The Metals, as Slats. Sewage and Industrial Wastes. 25(7)802-839.

Doyle, J.J., W.H. Pfander, S.E. Grebing, and J.O. Pierce, II. 1972. Effects of Dietary Cadmium on Growth and Tissue Levels in Sheep. Pages 181-186 in DD. Hemphill (Ed.). Trace Substances in Environmental Health - VI. Univ. of Missouri Ann. Conf., 6th Proc.

Dunning, J.B. 1984. Body Weights of 686 Species of North American Birds. Western Bird Banding Association. Monograph No. 1. Cave Creek, Arizona.

Eberhardt, L.E., W.C. Hanson, J.L. Bengtson, R.A. Garrott, and E.E. Hanson. 1982. Arctic Fox Home Range Characteristics in an Oil-Development Area. J. Wildl. Manage. 46(1):183-190.

Edwards, N.T. 1983. Polycyclic Aromatic Hydrocarbons (PAH's) in the Terrestrial Environment - A Review. J. Environ. Qual. 12:427-441. In: R. Eisler, 1987. Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. USFWS Biological Report 85(1.11) May 1987.

Eisler, R. 1985. Cadmium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service. Laurel, Maryland. Contaminant Hazards Reviews Report No. 2.

Eisler, R. 1987. Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service. Biological Report 85(1.11). May 1987.

Eisler, R. 1988. Lead Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Department of the Interior, Fish and Wildlife Service. Contaminant Hazard Reviews Report No. 14. Biological Report 85(1.14).

Eisler, R. 1993. Zinc Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. Biological Report 10. U.S. Fish and Wildlife Service. Laurel, Maryland.

Farrand, J., Jr. (Ed). 1983. The Audubon Society Master Guide to Birding, Volume 2. Alfred A. Knopf, New York, New York, 398 pp.

Foy, C.D., R.L. Chaney, and M.C. White. 1978. The Physiology of Metal Toxicity in Plants. Ann. Rev. Physiol. 29:511-566.

Gaur, J.P. 1988. Toxicity of Some Oil Constituents to *Selenastrum capricornutum*. Hydrobiol. 16(6):617-620. In: AQUIRE 1994.

Geiger, J.G. and A.L. Buikema, Jr. 1981. Oxygen Consumption and Filtering Rate of *Daphnia pulex* After Exposure to Water-Woluble Fractions of Naphthalene, Phenanthrene, No. 2 Fuel Oil and Coal-Tar Creosote. Bulletin of Environmental Contamination and Toxicology 27:783-789.

Gough, L.P., H.T. Shaklette, and A.A. Case. 1979. Element Concentrations Toxic to Plants, Animals and Man. U.S. Government Printing Office Washington, D.C. Geological Survey Bulletin 1466.

Haghiri, F. 1973. Cadmium Uptake in Plants. J. Environ. Qual. 2(1):93-96.

Harcharek, R. 1994. North Slope Borough 1993/1994 Economic Profile and Census Report. Department of Planning and Community Services, North Slope Borough, Barrow, Alaska.

Harding Lawson Associates. 1992. Offpost Operable Unit Endangerment Assessment/ Feasibility Study. Final Report. Technical support for Rocky Mountain Arsenal. 24 November 1992. Prepared for program Manager for Rocky Mountain Arsenal.

Hart Crowser. 1987. Environmental Assessment for North Warning System. Alaska.

Hartung, R. 1964. "Some Effects of Oils on Waterfowl." PhD Thesis. University of Michigan, Ann Arbor. In: R.C. Szaro, M.P. Dieter, G.H. Heinz, and J.F. Ferrell. 1978. Effects of Chronic Ingestion of South Louisiana Crude Oil on Mallard Ducklings. Environmental Research 17:426-436.

Hazardous Substance Data Bank (HSDB). 1994. National Institute of Health. Bethesda, Maryland.

Health Effects Assessment Summary Tables (HEAST). 1993. Environmental Criterion Assessment Office, U.S. Environmental Protection Agency, Cincinnati, Ohio.

Heath, J.S., K. Koblis, and S.L. Sager. 1993. Review of Chemical, Physical, and Toxicologic Properties of Components of Total Petroleum Hydrocarbons. J. of Soil Contamination. 2(i): 1-25.

Hedtke, S. and F.A. Puglisi. 1982. Short-Term Toxicity of Five Oils to Four Freshwater Species. Archives of Environmental Contamination and Toxicology 11:425-430.

Hensel, R., M.F. Arend, J. Thiele, P.O. McMilland, and S.V. Cuccarese. 1984. Living Resources of the Point Barrow, Oliktok Point and Boulder Creek Areas, Alaska: A Literature Survey. AEIDC. University of Alaska, Anchorage.

Hill, E.F. and M.B. Camardese. 1986. Lethal Dietary Toxicities of Environmental Contaminants and Pesticides to *Coturnix*. U.S. Fish and Wildlife Service. Technical Report 2. Washington, D.C. 147 p.

Hoffman, D.J., J.C. Franson, O.H. Pattee, C.M. Bunck, and A. Anderson. 1985. Survival, Growth and Accumulation of Ingested Lead in Nestling American Kestrels (*Falco Sparverius*). Arch. Environ. Contam. Toxicol. 14:89-94.

Hoffman D.J. and W.C. Eastin. 1981. TOXICOL LETT 6: 35-40. In: HSDB 1994.

Hull, R.N. and G.W. Suter II. 1994. Toxicological Benchmarks for Screening Contaminants of Potential Concern for Effects on Sediment Associated Biota: 1994 Revision. ORNL Environmental Restoration Program. ES/ER/TM-95/R1.

Integrated Risk Information System (IRIS). 1994. Environmental Criterion Assessment Office, U.S. Environmental Protection Agency, Cincinnati, Ohio.

Johnson, L. and B. Burns (Eds). 1984. Biology of the Arctic Char: Proceedings of the International Symposium on Arctic Char. University of Manitoba Press, Winnipeg, Man., Canada. 584 pp.

Kabata-Pendias, A. and H. Pendias. 1984. Trace Elements in Soil and Plants. CRC Press. Boca Raton, Florida.

Kauss, P.B. and T.C. Hutchinson. 1975. The Effects of Water-Soluble Petroleum Components on the Growth of Chlorella Vulgaris Beijerinck. Environmental Pollution (9):157-174.

Klaassen, C.D., M.O. Amdur, and J. Doull. 1986. Casarett and Doull's Toxicology, the Basic Science of Poisons. Third Edition. MacMillan Publishing Company. New York. 974 pp.

Klein, S.A. and D. Jenkins. 1983. The Toxicity of Jet Fuels to Fish - II. Water Research 17 (10):1213-1220.

Kostecki, P.T. and E.J. Calabrese. 1989. Petroleum Contaminated Soils. Volume 1. Lewis Publishers, Chelsea, Michigan. 357 pp.

Kraus, M.L. 1989. Bioaccumulation of Heavy Metals in Pre-Fledgling Tree Swallows, Tachycineta Bicolor. Bull. Environ. Contam. Toxicol. 43:407-414.

Lee, K. 1983. Vanadium in the Aquatic Ecosystem. Pages 155-187 in J.O. Nriagu, Aquatic Toxicology. John Wiley and Sons, New York, New York.

Lee, S.D. and L. Grant (Eds.). 1981. Health and Ecological Assessment of Polynuclear Aromatic Hydrocarbons. Pathotex Publ., Park Forest South, Illinois. In: Eisler, R. 1987. Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. USFWS Biological Report 85(1.11). May 1987.

Lehman, A.J. 1954. Appraisal of the Study of Chemicals in Food, Drugs, and Cosmetics. U.S. Association of Food and Drug Officials. 18:66.

Lewis, M.A., Evans, D.W., and J.G. Wiener. 1979. Manganese. In: R.V. Thurston, R.C. Russo, C.M. Ferrerolf, Jr., T.A. Edsall, and Y.M. Barber, Jr. (Eds.). 1979. A Review of the EPA Red Book: Quality Criteria for Water. American Fisheries Society. Bethesda, Maryland. pp. 137-144.

Lindsay, W.L. 1979. Chemical Equilibria in Soils. John Wiley and Sons, New York. 449 pages.

Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt (Eds). 1982. Research and Development Methods for Estimating Physio-Chemical Properties of Organic Compounds of Environmental Concern. Report No. C-82426 by Arthur D. Little, Inc., under contract DAMD-17-78-C-0873, U.S. Army Medical R&D Command. Fort Detrick, Maryland.

Mantel, N. and M.A. Schneiderman. 1975. Estimating Safe Levels: A Hazardous Undertaking. Cancer Res. 35:1379-1386.

Martin, A.C., H.S. Zim, and A.L. Nelson. 1961. American Wildlife and Plants: A Guide to Wildlife Food Habits. Dover Publications, New York. 500 pp.

Massachusetts Department of Environmental Protection (MDEP). 1993. Petroleum Policy: Development of Health-Based Alternative to the TPH Parameter. Prepared by ABB Environmental Services, Inc. Wakefield, Massachusetts. Project No. 06979-00. August.

Mautino, M. and J.U. Bell. 1987. Hematological Evaluation of Lead Intoxication in Mallards. Bull. Environ. Contam. Toxicol. 38:29-34.

MITRE. 1990. General Guidance for Ecological Risk Assessment at Air Force Installations. Prepared by the MITRE Corporation, Brooks Air Force Base, Texas (December 1990).

Moles, A., S.D. Rice, and S. Korn. 1979. Sensitivity of Alaskan Freshwater and Anadromous Fishes to Prudhoe Bay Crude Oil and Benzene. Transactions of the American Fisheries Society 108:408-414.

Nagy, K.A. 1987. Field Metabolic Rate and Food Requirement Scaling in Mammals and Birds. Ecol. Mono. 57:111-128.

National Academy of Sciences. 1980. Mineral Tolerance of Domestic Animals. Subcommittee on Mineral Toxicity in Animals. National Research Council. Washington, D.C.

National Oceanic and Atmospheric Administration (NOAA). August 1991. The Potential for Biological Effects of Sediment Sorbed Contaminants Tested in the National Status and Trends Program. Technical Memorandum NOS OMA 52. NOAA, Seattle, Washington.

National Petroleum Reserve in Alaska Task Force. 1978. 105(c) Land Use Study, Volume 2: Values and Resource Analysis. U.S. Department of the Interior. Anchorage, Alaska.

Nehring, R.B. and J.P. Goettl, Jr. 1974. Acute Toxicity of Zinc-Polluted Stream to Four Species of Salmonids. Bulleting of Environmental Contamination and Toxicology. 12(4):464-469.

Nowak, R.M. (Ed). 1991. Walker's Mammals of the World. Fifth Edition. Johns Hopkins University Press, Baltimore, Maryland.

O'Brien, J.W. 1978. Toxicity of Prudhoe Bay Crude Oil to Alaskan Arctic Zooplankton. Arctic 31(3):219-228.

Opresko, D.M., B.E. Sample, and G.W. Suter II. 1994. Toxicological Benchmarks for Wildlife: 1994 Revision. ORNL Environmental Restoration Program. ES/ER/TM-86/R1.

Palmer, R.S. (Ed). 1976. Handbook of North American Birds, Volume 2, pp. 244-273. Yale University Press, New Haven, Connecticut. 521 pp.

Patton, J.F. and M.P. Dieter. 1980. Effects of Petroleum Hydrocarbons on Hepatic Function in the Duck. Comp. Biochem. Physiol. 65C:33-36. In: R. Eisler, 1987. Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. USFWS Biological Report 85(1.11). May 1987.

Pickering, Q.H., E.P. Hunt, G.L. Phipps, R.H. Roush, W.E. Smith, D.L. Spehar, C.E. Stephens, and D.K. Tanner. 1983. Effects of Pollution on Freshwater Fish and Amphibians. JWPCF. 55(6):840-862.

Pitelka, F.A. 1959. Numbers, Breeding Schedule, and Territoriality in Pectoral Sandpipers of Northern Alaska. Condor 62(4)233-264.

Puls, R. 1988. Mineral Levels in Animal Health. Sherpa International. Clearbrook, British Columbia.

Radian Corporation. 1989. Environmental Impact Assessment for LIZ-3 Distant Early Warning Radar Station. Wainwright, Alaska.

Raven, P.H., R.H. Evert, and F.E. Eichorn. 1986. Biology of Plants. Worth Publishers, Inc. New York, New York. 775 pp.

Sax, I. and R.J. Lewis, Sr. 1989. Dangerous Properties of Industrial Materials. Seventh Edition. Van Nostrand, Reinhold, New York.

Schafer, E.W., Jr., W.A. Bowles, Jr., and J. Hurlbut. 1983. The Acute Oral Toxicity, Repellency, and Hazard Potential of 998 Chemicals to One or More Species of Wild and Domestic Birds. Arch. Environ Contam. Toxicol. 12:355-382.

Scott, S.L. 1983. Field Guide to the Birds. National Geographic Society. Washington, D.C. 464 pp.

Sims, R.C. and R. Overcash. 1983. Fate of Polynuclear Aromatic Compounds (PNAS) in Soil-Plant Systems. Residue Rev. 88:1-68. In: R. Eisler, 1987. Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. USFWS Biological Report 85(1.11). May 1987.

Skogland, T. 1980. Comparative Summer Feeding Strategies of Arctic and Alpine Rangifer. Journal of Animal Ecology. 49:81-98.

Snyder-Conn, E. 1994. Personal communication with E. Snyder-Conn, wildlife biologist with the U.S. Fish and Wildlife Service, Fairbanks, Alaska. Don Kellett, ICF Kaiser Engineers. 31 March 1994.

Soto, C., J. Hellebust, and T.C. Hutchinson. 1975. Effect of Napthalene and Aqueous Crude Oil Extracts on the Green Flagellate Chlamysomanas Angulosa. Canadian J. of Botany 53(2):118-126.

Spacie, A. and J.L. Hamelink. 1985. Bioaccumulation, Chapter 17, pp. 495-525 in Fundamentals of Aquatic Toxicology. G.M. Rand and S.R. Petrocelli (Eds). Hemisphere Publishing Corporation, New York.

Suter, G.W and J.B. Mabrey. 1994. Toxicological Benchmarks for Screening Potential Contaminants of Concern for Effects on Aquatic Biota: 1994 Revision. Environmental Sciences Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee.

Szaro, R.C. and P.H. Albers. 1977. Effects of External Applications of No. 2 Fuel Oil on Common Eider Eggs. Pp. 164-167. In: D.A. Wolfe (Ed). Fate and Effects of Petroleum Hydrocarbons in Marine Ecosystems and Organisms. Pergamon Press, New York.

Szaro, R.C., M.P. Dieter, G.H. Heinz, and J.F. Ferrell. 1978. Effects of Chronic Ingestion of South Louisiana Crude Oil on Mallard Ducklings. Environmental Research 17:426-436.

Talmadge, S.S. and B.T. Walton. 1991. Small Mammals as Monitors of Environmental Contaminants. Reviews of Environ. Contam. Toxicol. 119:47-145.

Travis, C.C. and A.D. Arms. 1988. Bioconcentration of Organics in Beef, Milk, and Vegetation. Environ. Sci. Technol. 22(3):271-274.

Traynor, M.F. and B.D. Knezek. 1973. Effects of Nickel and Cadmium Contaminated Soils on Nutrient Composition of Corn Plants. Pages 82-87 in D.D. Hemphill (Ed.), Trace Substances in Environmental Health - VII. University of Missouri Annual Conference, 7th Proceedings.

U.S. Army Corps of Engineers (USACOE). 1991. Baseline Risk Assessment for Eight Selected Study Areas at Aberdeen Proving Ground. Appendix C, Draft Document.

U.S. Air Force. 1991. Handbook to Support the Installation Restoration Program (IRP) Statements of Work. Volume 1, Remedial Investigation/Feasibility Studies (RI/FS). Human Systems Division, Brooks Air Force Base, Texas.

U.S. Air Force. 1993a. Work Plan for Remedial Investigations/Feasibility Studies at Air Force Radar Stations. Final Draft. Prepared for USAF Center for Environmental Excellence, Environmental Restoration Program Office, Brooks AFB, Texas. Prepared by ICF Technology Incorporated.

U.S. Air Force. 1993b. Sampling and Analysis Plan for DEW Line and Cape Lisburne Radar Stations. Prepared for USAF Center for Environmental Excellence, Environmental Restoration Program Office, Brooks AFB, Texas. Prepared by ICF Technology Incorporated.

U.S. Air Force. 1996. Remedial Investigation and Feasibility Study, Wainwright Radar Installation, Alaska. Final Draft. Prepared for USAF Center for Environmental Excellence, Environmental Restoration Program Office, Brooks AFB, Texas. Prepared by ICF Technology Incorporated.

U.S. Environmental Protection Agency (EPA). 1976. Quality Criteria for Water. Office of Water and Hazardous Materials. Washington, D.C.

U.S. Environmental Protection Agency (EPA). 1980. Ambient Water Quality Criteria for Polynuclear Aromatic Hydrocarbons. EPA 440/5-80-069. In: R. Eisler, 1987. Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. USFWS Biological Report 85(1.11). May 1987.

U.S. Environmental Protection Agency (EPA). 1985. Environmental Profiles and Hazard Indices for Constituents of Municipal Sludge: Iron. Office of Water Regulations and Standards. Washington, D.C.

U.S. Environmental Protection Agency (EPA). 1986a. Guidelines for Carcinogen Risk Assessment. Federal Register 51:33992-34013.

U.S. Environmental Protection Agency (EPA). 1986b. Guidelines for the Health Risk Assessment of Chemical Mixtures. Federal Register 51:34014-34025.

U.S. Environmental Protection Agency (EPA). 1986c. Quality Criteria for Water. Office of Water Regulations and Standards. Washington D.C. EPA 440/5-86-001.

U.S. Environmental Protection Agency (EPA). 1987. Ambient Water Quality Criteria for Zinc - 1987. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-87-003. P887-153581.

U.S. Environmental Protection Agency (EPA). 1988. Ambient Water Quality Criteria for Aluminum. Office of Water Regulations and Standards, Criteria and Standards Division. Washington, D.C. EPA 440/5-86-008.

U.S. Environmental Protection Agency (EPA). 1989a. Risk Assessment Guidance for Superfund, Human Health Evaluation Manual, Part A. Office of Solid Waste and Emergency Response. Washington, D.C.

U.S. Environmental Protection Agency (EPA). 1989b. Risk Assessment Guidance for Superfund: Volume 2, Environmental Evaluation Manual. Office of Solid Waste and Emergency Response. Washington, D.C.

U.S. Environmental Protection Agency (EPA). 1989c. Exposure Factors Handbook. Office of Health and Environmental Assessment. U.S. Environmental Protection Agency, Washington, D.C. EPA/600/8-89/041.

U.S. Environmental Protection Agency (EPA). 1991a. Region 10 Supplemental Risk Assessment Guidance for Superfund. Seattle, Washington. 16 August 1991.

U.S. Environmental Protection Agency (EPA). 1991b. Role of the Baseline Risk Assessment in Superfund Remedy Selection Decisions. Office of Solid Waste and Emergency Response. Washington, D.C. 22 April 1991.

U.S. Environmental Protection Agency (EPA). 1991c. Human Health Evaluation Manual, Part B: Development of Risk-Based Preliminary Remediation Goals. Office of Solid Waste and Emergency Response, U.S. Environmental Protection Agency, Washington, D.C. December 13, 1991.

U.S. Environmental Protection Agency (EPA). 1992a. Framework for Ecological Risk Assessment. EPA/630/R-92/001. NTIS #PB93-102192. Washington, D.C. February 1992.

U.S. Environmental Protection Agency (EPA). 1992b. Oral Reference Doses and Oral Slope Factors for JP-4, JP-5, Diesel fuel, and Gasoline. Environmental Criterion Assessment Office, Office of Research and Development, U.S. Environmental Projection Agency, Cincinnati, Ohio. March 24, 1992.

U.S. Environmental Protection Agency (EPA). 1992c. Superfund Health Risk Technical Support Center. Environmental Criteria and Assessment Office, Cincinnati, Ohio.

U.S. Environmental Protection Agency (EPA). 1994. Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments. EPA Environmental Response Team, Edison, New Jersey. September 26, 1994. Review Draft.

U.S. Environmental Protection Agency (EPA). 1995a. Remedial Investigation and Feasibility Study, Wainwright Radar Installation, Alaska. Final Draft. Prepared by ICF Technology Incorporated.

U.S. Fish and Wildlife Service. 1982. Arctic National Wildlife Refuge Coastal Plain Resource Assessment-Initial Report. Baseline Study of Fish, Wildlife and their Habitats. U.S. Department of the Interior, Anchorage Alaska.

U.S. Fish and Wildlife Service. 1986. Lethal Dietary Toxicities of Environmental Contaminants and Pesticides to Coturnix. Fish and Wildlife Technical Report 2. Washington D.C. Hill, E.F. and M.B. Camardese.

U.S. Geological Survey (USGS). 1955 (Minor Revisions 1982). Wainwright (C-2) Quadrangle, Alaska-North Slope Borough, 1:63,360 Series (Topographic).

U.S. Geological Survey (USGS). 1985. Study and Interpretation of the Chemical Characteristics of Natural Water. USGS Water-Supply Paper 2254. Third Edition.

University of Alaska, Arctic Environmental Information and Data Center. 1978. Kaktovik, pp 2-29.

Veith et al. 1979. In: A. Spacie and J.L. Hamelink. 1985. Bioaccumulation, Chapter 17, pp. 495-525 in Fundamentals of Aquatic Toxicology. G.M. Rand and S.R. Petrocelli (Eds). Hemisphere Publishing. New York.

Verschueren, K. 1983. Handbook of Environmental Data of Organic Chemicals. Second Edition. New York: Van Nostrand Reinhold Co. p. 629

Von Burg, R. 1993. Evaluation of TPH as a Determinant for Petroleum Hydrocarbon Cleanup in Soil. ICF Kaiser Engineers, Oakland, California.

Walker, D.A., P.J. Webber, K.R. Everett, and J. Brown. 1978. Effects of Crude and Diesel Oil Spills on Plant Communities at Prudhoe Bay, Alaska, and the Derivation of Oil Spill Sensitivity Maps. Arctic 31(3):242-259.

Wang, D.T. and O. Meresz. 1982. Occurrence and Potential Uptake of Polynuclear Aromatic Hydrocarbons of Highway Traffic Origin by Proximally Grown Food Crops. In: R. Eisler, 1987. Polycyclic Aromatic Hydrocarbon Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. USFWS Biological Report 85(1.11). May 1987.

Weeks, J.A., G.H. Drendel, R.S. Jagan, T.E. McManus, and P.J. Sczerzenie. 1988. Diesel Oil and Kerosene Background Statement. Prepared by Labat-Anderson, Inc. for the U.S. Department of Agriculture, Forest Service. February.

White, R.G. and J. Trudell. 1980. Habitat Preference and Forage Consumption by Reindeer and Caribou Near Atkasook, Alaska. Arctic and Alpine Research 12(4):511-529.

Wong, C.K., F.R. Engelhardt, and J.R. Strickler. 1981. Survival and Fecundity of *Daphnia pulex* on Exposure to Particulate Oil.

Woodward-Clyde Corporation. 1993. Natural Resources Plan: North Coastal Long Range Radar Sites. Final Draft. Prepared for the United States Air Force.

Wootton, R.J. 1976. The Biology of the Sticklebacks. Academic Press, New York. 387 pp.

APPENDIX A

RISK CHARACTERIZATION SPREADSHEETS

TABLE A-1. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion
Endpoint: Noncancer
Assumptions: Site-specific
Installation: Wainwright
Site: Diesel Fuel Spills (SS04)
File: SS04SONC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	14	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	0.000001	0.000001	0.000001
Body Weight	(kg)	70	70	15
Averaging Time	(ED x 365 days/year)	3,650	17,885	2,190

Chemical	Oral RfD	Concentration Soil (mg/kg)	ADD by Receptor Group (mg/kg-day)			Hazard Quotients	
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult/Child
DRPH	0.08	4,900	1.34e-04	5.75e-04	5.37e-03	1.68e-03	7.43e-02
GRPH	0.2	120	3.29e-06	1.41e-05	1.32e-04	1.64e-05	7.28e-04
					HAZARD INDEX	0.002	0.075

TABLE A-2. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route:	Soil Ingestion
Endpoint:	Cancer
Assumptions:	Site-specific
Installation:	Wainwright
Site:	Diesel Fuel Spills (SS04)
File:	SS04SOCA.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	14	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	0.000001	0.000001	0.000001
Body Weight	(kg)	70	70	15
Averaging Time	(lifetime in days)	25,550	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Soil (mg/kg)	LADD by Receptor Group (mg/kg-day)			Cancer Risk	
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult/Child
GRPH	0.0017	120	4.70e-07	9.86e-06	1.13e-05	7.98e-10	3.59e-08
					CANCER RISK	8e-10	4e-08

TABLE A-3. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion
Endpoint: Noncancer
Assumptions: Site-specific
Installation: Wainwright
Site: Landfill (LF05)
File: LF05SONC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	14	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	0.000001	0.000001	0.000001
Body Weight	(kg)	70	70	15
Averaging Time	(ED x 365 days/year)	3,650	17,885	2,190

Chemical	Oral RfD	Concentration Soil (mg/kg)	ADD by Receptor Group (mg/kg-day)			Hazard Quotients	
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult/Child
Cadmium	0.001	72	1.97e-6	8.45e-6	7.89e-5	2.00e-3	1.60e-2
GRPH	0.2	200	5.48e-06	2.35e-05	2.19e-04	2.74e-05	1.21e-03
					HAZARD INDEX	0.002	0.02

TABLE A-4. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion
Endpoint: Cancer
Assumptions: Site-specific
Installation: Wainwright
Site: Landfill (LF05)
File: LF05SOCA.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	14	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	0.000001	0.000001	0.000001
Body Weight	(kg)	70	70	15
Averaging Time	(lifetime in days)	25,550	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Soil (mg/kg)	LADD by Receptor Group (mg/kg-day)			Cancer Risk	
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult/Child
GRPH	0.0017	200	7.83e-07	1.64e-05	1.88e-05	1.33e-09	5.99e-08
					CANCER RISK	1e-09	6e-08

TABLE A-5. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
Endpoint: Cancer
Assumptions: Site-specific
Installation: Wainwright
Site: Landfill (LF05)
File: LF05WACA.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Water Ingestion	(L/day)	2	2	NA
Exposure Frequency	(days/year)	14	180	NA
Exposure Duration	(years)	10	55	NA
Conversion Factor	(kg/mg)	1	1	NA
Body Weight	(kg)	70	70	NA
Averaging Time	(lifetime in days)	25,550	20,075	NA

Chemical	Carcinogen Oral Slope Factor	Concentration Water (mg/L)	LADD by Receptor Group (mg/kg-day)			Cancer Risk		
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult	Native Northern Child
1,2-Dichloroethane	0.091	0.0062	9.71e-07	8.74e-05	NA	8.83e-08	7.95e-06	NA
					CANCER RISK	9e-08	8e-06	0

TABLE A-6. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion
Endpoint: Noncancer
Assumptions: Site-specific
Installation: Wainwright
Site: Garage (SS07)
File: SS07SONC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	14	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	0.000001	0.000001	0.000001
Body Weight	(kg)	70	70	15
Averaging Time	(ED x 365 days/year)	3,650	17,885	2,190

Chemical	Oral RfD	Concentration Soil (mg/kg)	ADD by Receptor Group (mg/kg-day)			Hazard Quotients	
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult/Child
DRPH	0.08	120,000	3.29e-03	1.41e-02	1.32e-01	4.11e-02	1.82e+00
GRPH	0.2	120	3.29e-06	1.41e-05	1.32e-04	1.64e-05	7.28e-04
RRPH	0.08	77,000	2.11e-03	9.04e-03	8.44e-02	2.64e-02	1.17e+00
Tetrachloroethene	0.01	11.5	3.15e-07	1.35e-06	1.26e-05	3.15e-05	1.40e-03
					HAZARD INDEX	0.068	2.990

TABLE A-7. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Soil Ingestion
Endpoint: Cancer
Assumptions: Site-specific
Installation: Wainwright
Site: Garage (SS07)
File: SS07SOCA.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Soil Ingestion Rate	(mg/day)	50	100	200
Exposure Frequency	(days/year)	14	30	30
Exposure Duration	(years)	10	49	6
Conversion Factor	(kg/mg)	0.000001	0.000001	0.000001
Body Weight	(kg)	70	70	15
Averaging Time	(lifetime in days)	25,550	25,550	25,550

Chemical	Carcinogen Oral Slope Factor	Concentration Soil (mg/kg)	LADD by Receptor Group (mg/kg-day)			Cancer Risk	
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult/Child
GRPH	0.0017	120	4.70e-07	9.86e-06	1.13e-05	7.98e-10	3.59e-08
Tetrachloroethene	0.052	11.5	4.50e-08	9.45e-07	1.08e-06	2.34e-09	1.05e-07
					CANCER RISK	3e-09	1e-07

TABLE A-8. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
Endpoint: Noncancer
Assumptions: Site-specific
Installation: Wainwright
Site: Garage (SS07)
File: SS07WANC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Water Ingestion	(L/day)	2	2	NA
Exposure Frequency	(days/year)	14	180	NA
Exposure Duration	(years)	10	55	NA
Conversion Factor	(kg/mg)	1	1	NA
Body Weight	(kg)	70	70	NA
Averaging Time	(ED x 365 days/year)	3,650	20,075	NA

Chemical	Oral RfD	Concentration Water (mg/L)	ADD by Receptor Group (mg/kg-day)			Hazard Quotient		
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult	Native Northern Child
bis-(2Ethylhexyl) Phthalate	0.02	0.016	1.75e-05	2.25e-04	NA	8.77e-04	1.13e-02	NA
					HAZARD INDEX	8.77e-04	1.13e-02	0.00

TABLE A-9. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
Endpoint: Cancer
Assumptions: Site-specific
Installation: Wainwright
Site: Garage (SS07)
File: SS07WACA.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Water Ingestion	(L/day)	2	2	NA
Exposure Frequency	(days/year)	14	180	NA
Exposure Duration	(years)	10	55	NA
Conversion Factor	(kg/mg)	1	1	NA
Body Weight	(kg)	70	70	NA
Averaging Time	(lifetime in days)	25,550	20,075	NA

Chemical	Carcinogen Oral Slope Factor	Concentration Water (mg/L)	LADD by Receptor Group (mg/kg-day)			Cancer Risk		
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult	Native Northern Child
bis-(2Ethylhexyl) Phthalate	0.014	0.016	2.50e-06	2.25e-04	NA	3.51e-08	3.16e-06	NA
1,2-Dichloroethane	0.091	0.0018	2.82e-07	2.54e-05	NA	2.56e-08	2.31e-06	NA
					CANCER RISK	6e-08	5e-06	0

TABLE A-10. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
Endpoint: Noncancer
Assumptions: Site-specific
Installation: Wainwright
Site: Vehicle Storage Area (SS09)
File: SS09WANC.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Water Ingestion	(L/day)	2	2	NA
Exposure Frequency	(days/year)	14	180	NA
Exposure Duration	(years)	10	55	NA
Conversion Factor	(kg/mg)	1	1	NA
Body Weight	(kg)	70	70	NA
Averaging Time	(ED x 365 days/year)	3,650	20,075	NA

Chemical	Oral RfD	Concentration Water (mg/L)	ADD by Receptor Group (mg/kg-day)			Hazard Quotient		
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult	Native Northern Child
Barium	0.07	0.75	8.22e-04	1.06e-02	NA	1.17e-02	1.51e-01	NA
Manganese	0.005	3.8	4.16e-03	5.35e-02	NA	8.33e-01	1.07e+01	NA
Vanadium	0.007	0.063	6.90e-05	8.88e-04	NA	9.86e-03	1.27e-01	NA
Zinc	0.3	3.3	3.62e-03	4.65e-02	NA	1.21e-02	1.55e-01	NA
					HAZARD INDEX	8.67e-01	1.11e+01	0.00

TABLE A-11. DEW LINE INSTALLATION RISK ASSESSMENT SPREADSHEET

Route: Water Ingestion
Endpoint: Cancer
Assumptions: Site-specific
Installation: Wainwright
Site: Vehicle Storage Area (SS09)
File: SS09WACA.WK1

Exposure Assumptions		DEW Line Worker	Native Northern Adult	Native Northern Child
Water Ingestion	(L/day)	2	2	NA
Exposure Frequency	(days/year)	14	180	NA
Exposure Duration	(years)	10	55	NA
Conversion Factor	(kg/mg)	1	1	NA
Body Weight	(kg)	70	70	NA
Averaging Time	(lifetime in days)	25,550	20,075	NA

Chemical	Carcinogen Oral Slope Factor	Concentration Water (mg/L)	LADD by Receptor Group (mg/kg-day)			Cancer Risk		
			DEW Line Worker	Native Northern Adult	Native Northern Child	DEW Line Worker	Native Northern Adult	Native Northern Child
1,2-Dichloroethane	0.091	0.0016	2.50e-07	2.25e-05	NA	2.28e-08	2.05e-06	NA
					CANCER RISK	2e-08	2e-06	0

APPENDIX B

TOXICITY PROFILES

TOXICOLOGY PROFILE FOR DIESEL FUEL (DRPH)

GENERAL DATA

Petroleum fuels are classified into light, middle, and heavy distillate fuels. Gasoline is a typical light distillate fuel while diesel fuel is considered to be a middle distillate material obtained from the distillation of crude oil. Included in this category of middle distillate fuels are jet fuel, kerosene, and #2 fuel oils. Many of the ecological and toxicological effects of these materials are very similar.

The chemical composition of diesel fuel is extremely variable and depends upon the crude oil source, types of processing and refining, blending, and additives employed. These fuels are formulated to meet physical characteristics and not a specific chemical composition. Viscosity and volatility are the principal determinants of the fuel specifications. Diesel #1 is primarily a kerosene type of fuel and produced mainly from straight run middle distillates. Diesel #2 also contains straight run middle distillate but is also blended with straight run kerosene, straight run gas oils, light vacuum distillate, and light thermally and/or catalytically cracked streams (IARC 1989).

Like other petroleum derived fuels, diesel fuels consist of paraffins, olefins, cycloparaffins, isoparaffins, and aromatics as well as additives. Additives can include amyl nitrates, alcohols, n-hexyl nitrate, and octyl nitrate at levels of 0.1 - 0.2 % (Kirk-Othmer 1984). The total aromatic content of diesel fuel is also variable but levels between 23 - 38% have been reported. The average total aromatic is probably in the range of 25%. The concentrations of the principal aromatic species of toxicological significance is presented in Table B-1.

TABLE B-1. REPRESENTATIVE VALUES FOR TOXICOLOGICALLY SIGNIFICANT AROMATIC CONTENT FOR DIESEL FUEL# 2.

COMPONENT	APPROXIMATE CONCENTRATION
Benzene	< 50 ppm with an average of 10 ppm
Ethylbenzene	300 ppm
Toluene	200 ppm (max)
Xylene (mixed)	2400 ppm

(personal communication, Chevron Corp.)

The odor threshold of diesel fuel is approximately 0.8 ppm.

FATE AND TRANSPORT

Microbial degradation, plus evaporation, can remove up to 90% of the added diesel fuel to soil. Depending on the soil characteristics, the half-life of diesel fuel in soil ranges for 1 - 8 weeks (Song 1988). Volatilization to the air occurs and diesel fuel can be detected by its odor in the air. However, a vapor pressure value could not be located in the literature. Diesel fuel will percolate through the soil and float on the ground water. When spilled onto surface water, diesel fuels can be toxic to fish, waterfowl and algae.

TOXICITY DATA

Human Toxicological Profile

Like other solvents, diesel fuel can be expected to be a central nervous system (CNS) depressant. However, since this fuel is not as volatile as gasoline, breathing vapors at concentrations sufficient to achieve a level of intoxication is not likely at normal temperatures and pressures. An attempt to generate a kerosene (diesel) laden atmosphere only resulted in an ambient concentration of 14 ppm (Carpenter et al. 1976). However, under certain occupational settings like tank cleaning, it may be possible to generate mists or aerosols that can lead to symptoms of overexposure. As with kerosene, these symptoms may include headache, dizziness, weakness, confusion, drowsiness and possibly death (HSDB 1991).

Ingestion of diesel fuel can occur during siphoning, abuse situations, or from contaminated well-water. Ingestion may be accompanied by a burning sensation in the mouth, pharynx and chest, gastrointestinal hypermotility and diarrhea (Gosselin et al. 1984), and possibly nausea and vomiting. A serious complication is the aspiration of hydrocarbons into the lung which produces a potentially-lethal hemorrhagic pneumonitis (Lee and Seymour 1979).

There have been reports of acute renal failure following persons exposed to diesel fuel (Barrientos et al. 1977; Crisp et al. 1979). Kryzanovskij (1971) reports that workers cleaning diesel storage tanks have an increased incidence of disease in general, and specifically cardiovascular disease and bronchitis over control shipyard workers.

Animal Toxicology and Significant Studies

The acute oral and dermal LD_{50} of diesel fuel is in the range of 9 ml/kg body weight. Eye irritation properties were minimal, but the primary skin irritation score of a marketplace sample was 6.8 indicating that this material is a strong skin irritant (Beck et al. 1982) Chronic skin contact can be expected to produce defatting, fissuring and cracking. There are no readily available reports on hypersensitivity response to diesel fuels can be expected to occur since products on either side of diesel fuels distillation range have been reported to produce hypersensitivity reactions (Beck et al. 1982). Dermal absorption of gasoline is unlikely to result in systemic toxicity, but chronic poisoning of the readily absorbable alkyl lead additives is possible.

Exposure of CD-1 mice to diesel vapor for 8 hrs per day on 5 consecutive days resulted in a decrement of performance on the roto-rod test, square box activity test and hot plate test. However, the corneal reflex and inclined plane test was unaffected. General observations noted vasodilation, ataxia, poor grooming and in some cases tremor (Kainz and White 1982).

Exposure of rats to aerosolized diesel fuel at concentrations up to 6 mg/L produced direct toxic effects on the lungs but did not produce any neurotoxicity (Dalbey et al. 1987).

Reproductive Toxicity

Female rats were exposed 6 hours per day to air concentrations of 0, 100, and 400 ppm during days 6 through 15 of gestation. Neither jet fuel or number 2 fuel oil produced any significant detrimental effects on the reproductive parameters of the experimental animals (Beliles and Mecler 1982). Neither Jet Fuel A or diesel fuel at exposure levels of 400 ppm, 6 hrs per day, 5 day per week for 8 weeks reduced the fertility of CD-1 male mice (API 1980a,b).

Genotoxicity

Kerosene, jet fuel and diesel fuel all tested negatively in the standard Ames bioassay. However, the "Modified Ames Assay" (Blackburn et al. 1988) on two straight run gas oils did demonstrate mutagenicity. (Straight run gas oil can be considered similar to diesel oils.) Diesel fuel was also negative in the mouse lymphoma assay but positive on the rat bone marrow cytogenetics assay when administered by intraperitoneal injection (Conaway et al. 1982). Heating oil #2 produced a positive Ames test as well as positive results in two other short term bioassays (Rothman and Emmett 1988). Dominant lethal testing of Jet fuel A and diesel fuel was negative at 400 ppm to male CD-1 mice (API 1980a,b).

Carcinogenicity

In a classical mouse skin painting bioassay, all petroleum fractions derived from a crude oil source that boiled between 120 and 700°F showed a low level of tumorigenic activity (Lewis et al. 1982). Home heating oil also showed a low level of tumorigenicity in a more recent mouse skin painting assay (Witschi et al. 1987).

In a case referent study, Seimiatycki et al. (1987) reported an increase of several specific cancers associated with exposures to different petroleum products. Leaded gasoline was associated with stomach cancer; aviation gasoline with kidney cancer; diesel fuel with non adenocarcinoma of the lung and prostate cancer and mineral spirits with squamous cell lung cancer. However not all parameters of concern were properly controlled, excluded or assessed making conclusions from this study inappropriate.

IARC (1989) has classified diesel fuel as having limited evidence of carcinogenicity in animals. Light diesel fuels are not classifiable as to their carcinogenicity to humans (Group 3).

REGULATIONS AND STANDARDS

Neither the American Conference of Governmental Industrial Hygienists (ACGIH) nor OSHA have recommended or established permissible exposure standards (PELs) for diesel fuels. However, NIOSH, has recommended a 10 hour time-weighted average of 100 mg/m^3 or 14 ppm for kerosene (NIOSH 1977). Because of the complexity and variability in composition, OSHA regulates the toxic components by their respective PELs (i.e., n-hexane, benzene, etc.).

Diesel fuels, as such, are not mentioned in HEAST (1990) nor identified for a specific cancer Potency Factor (CPF) or reference dose (RfD). However, individual components such as benzene, other aromatics and for n-hexane having CPF or RfD values should be evaluated by themselves.

REFERENCES

API. 1980a. Mutagenicity Evaluation of Jet Fuel A in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-03. American Petroleum Institute Medical Research Publications, Washington, D.C.

API. 1980b. Mutagenicity Evaluation of Diesel Fuel in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-04. American Petroleum Institute Medical Research Publications, Washington, D.C.

Barrientos, A., M.T. Ortuno, and J.M Morales et al. 1977. Acute Renal Failure After Use of Diesel Fuel as a Shampoo. Arch.Intern. Med. 137:1217-1219.

Beck, L.S., D.I. Hepler, and K.L. Hansen. 1982. The Acute Toxicology of Selected Petroleum Hydrocarbons. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.

Beliles, R.P. and F.J. Mecler. 1982. Inhalation Teratology of Jet Fuel, Fuel Oil and Petroleum Naphtha in Rats. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.

Blackburn, G.R., R.A. Deitch, and T.A. Roy et al. 1988. Estimation of the Dermal Carcinogenic Potency of Petroleum Fractions Using a Modified Ames Assay. In: Polynuclear Aromatic Hydrocarbons: A Decade of Progress, Proceedings of the Tenth International Symposium. M. Cooke and A.J. Dennis, editors. Battelle Press, Columbus Ohio, 99 83-97.

Carpenter, C.P., D.L. Geary, and R.C. Meyers et al. 1976. Petroleum Hydrocarbon Toxicity Studies XI. Animal and Human Response to Vapors of Deodorized Kerosene. Toxicol. Appl. Pharmacol. 36:443-456.

Conaway, C.C., C.A. Schreiner, and S.T. Cragg. 1982. Mutagenicity Evaluation of Petroleum Hydrocarbons. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.

Crisp, A.J., A.K. Bhalla, and B.I. Hoffbrand. 1979. Acute Tubular Necrosis After Exposure to Diesel Oil. Brit. Med J. 2:177-178.

Dalbey, W., M. Henry, and R. Holmberg et al. 1987. Role of Exposure Parameters in the Toxicity of Aerosolized Diesel Fuel in the Rat. J. Appl. Toxicol. 7:265-275.

Gosselin, R.E., R.P. Smith, and H.C. Hodge. 1984. Clinical Toxicology of Commercial Products. 5th Ed. Baltimore, Maryland: Williams & Wilkins.

HEAST. 1990. Health Effects Assessment Summary Tables, Office of Emergency and Remedial Response, Environmental Protection Agency. OERR 9200 6-303, (90-4).

HSDB. 1991. Hazardous Substances Data Bank. National Library of Medicine. Washington, D.C.

IARC. 1989. Monographs on the Evaluation of Carcinogenic Risks to Humans. Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels. International Agency for Research on Cancer, Lyon France. Vol 45.

Kainz, R.J. and L.E. White. 1982. Consequences Associated with the Inhalation of Uncombusted Diesel Vapor. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.

Kirk Othmer. 1984. Diesel Fuel. In: Kirk-Othmer Encyclopedia of Chemical Technology. F. Mark, M. Grayson, and D. Eckroth et al., Eds. 3rd Ed. John Wiley and Sons, New York.

Kryzanovskij, N.V. 1971. Occupational Health Conditions Associated with the Cleaning of Oil Tankers and Their Effect on Worker's Health. Gig. Tr. Prof. Zabol. 15:14-17.

Lee T. and W. Seymour. 1979. Pneumonitis Caused by Petrol Siphoning. (Letter). The Lancet 8134:149.

Lewis, S.C., R.W. King, S.T. Cragg, and D.W. Hillman. 1982. Skin Carcinogenic Potential of Petroleum Hydrocarbons. 2. Carcinogenesis of Crude Oil, Distillate Fractions and Chemical Class Subfractions. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.

NIOSH. 1977. Criteria for a Recommended Standard...Occupational Exposure to Refined Petroleum Solvents. National Institute for Occupational Safety and Health, Cincinnati, Ohio. NIOSH Publication #77-192.

Rothman, N. and E.A. Emmett. 1988. The Carcinogenic Potential of Selected Petroleum-derived Products. Occup. Med.: State Art Rev. 3:475-482.

Siemiatycki, J., R. Dewar, and L. Nadon et al. 1987. Associations Between Several Sites of Cancer and Twelve Petroleum Derived Liquids. Scan. J. Work Environ. Health. 13:493-504.

Song, H.G. 1988. Petroleum Hydrocarbons in Soil: Biodegradation and Effects on the Microbial Community. Ph.D. Thesis, University of Medicine and Dentistry of New Jersey.

Witschi, H.P., L.H. Smith, and E.L. Frome et al. 1987. Skin Tumorigenic Potential of Crude and Refined Coal Liquids and Analogous Petroleum Products. Fund. Appl. Toxicol. 9:297-303.

TOXICOLOGY PROFILE FOR GASOLINE (GRPH)

GENERAL DATA

The chemical composition of gasoline is extremely variable, depending upon the crude oil starting material, types of processing and refining, blending and additives employed. Gasolines are formulated to meet fuel performance specifications, not to achieve a specific chemical composition. Volatility must be within a certain range to avoid vapor lock (too high) or sluggish acceleration (too low). In addition, the air-fuel mixture within the cylinder must burn uniformly to prevent "pinging" or "knocking." Often small quantities of butanes, pentanes, organo lead compounds or branched chain hydrocarbons are added to achieve uniform burning rates. McDermott and Killiany (1978) published a detailed gas chromatographic analysis of a premium grade gasoline listing 21 components which accounted for 92% of the gasoline vapors (Table B-2). Low-volatility hydrocarbons (high carbon numbers) were not well represented.

Gasoline additives include organic lead (tetraethyl lead and tetramethyl lead) to a concentration of 0.1 g/gallon (7 ppm). Alkyl lead vapors have low volatility (vapor pressure = 0.4 mm Hg) compared to gasoline (400-775 mm Hg), so lead compounds should not be acutely hazardous by inhalation. To prevent accumulation of lead deposits, scavenging agents are added to fuels: ethylene dichloride (EDC) and ethylene dibromide (EDB), usually in a molar ratio EDC/EDB/Pb = 2:2:1.

FATE AND TRANSPORT

Gasoline released into the environment would be expected to evaporate rapidly due to its high vapor pressure (400-477 mm Hg). Studies of gasoline fate when added to soils show that the main clearance mechanism was evaporation which can account for up to 75% removal from surface soils (Donaldson 1990). Microbial degradation, plus evaporation, can remove up to 90% of the added gasoline (Song 1988). Benzene, a volatile gasoline component of major toxicological interest, has a half life in the air of less than 1 day (Korte and Klein 1982). Gasoline has appreciable water solubility (12-16%) so it would be transported in ground water and may be found in well water.

TOXICITY DATA

Human Toxicological Profile

Like other solvents, gasoline has potent central nervous system (CNS) depressant activity. Breathing vapors at concentrations achieved during "huffing" or occupational overexposures has led to a variety of neurological symptoms: hallucinations, encephalopathy, ataxia, convulsions, Tourette's Disease, vertigo and nystagmus and peripheral neuropathy (Von Burg 1989). Many of these symptoms may be attributed to n-hexane or alkyl lead compounds.

TABLE B-2. COMPOSITION OF A PREMIUM-GRADE GASOLINE

COMPOUND	VOL %
Propane	0.8
n-Butane	38.1
Isobutane	5.2
n-Pentane	7.0
Cyclopentane	0.7
2,3-DM-butane	0.7
2-M-pentane	2.1
3-M-pentane	1.6
n-Hexane	1.5
M-cyclopentane	1.3
2,4-DM-pentane	0.4
2,3-DM-pentane	0.7
2,2,4-TM-pentane	0.5
Isobutylene	1.1
2-M-1-butane	1.6
c-2-pentene	1.2
2-M-2-butene	1.7
Benzene	0.7
Toluene	1.8
Xylene (m,p,o)	0.5
Total %	92.1

Ingestion of gasoline can occur during siphoning, abuse situations, or from contaminated well-water. Ingestion is accompanied by a burning sensation in the mouth, pharynx and chest. Swallowing large amounts of gasoline leads to coma and death by respiratory depression. A serious complication is the aspiration of hydrocarbons into the lung which produces a potentially-lethal hemorrhagic pneumonitis (Lee and Seymour 1979).

Inhalation exposure to gasoline at concentrations of 1,000 - 5,000 ppm for 15 - 60 minutes can produce CNS depression. A 5-minute exposure to 20,000 ppm (20%) has been reported to be fatal (Von Burg 1989).

Three epidemiologic studies of refinery workers showed no increased cancer risk in refinery workers (Hanis et al. 1982; Kaplan 1986; Wong 1987). In an epidemiological study of refinery workers and gasoline handlers, Thomas et al. (1982) found a significant increase in stomach and brain cancer with a trend to increased leukemia and cancer of the skin, prostate and pancreas.

Animal Toxicology and Significant Studies

The acute dermal LD_{50} of gasoline in rabbits is reported to be <5 ml/kg (Von Burg 1989). Liquid gasoline is considered a primary skin irritant because of the defatting and fissuring which occurs upon repeated contact. Hypersensitivity response to gasoline can occur. Dermal absorption of gasoline is unlikely to result in systemic toxicity, but chronic poisoning of the readily absorbable alkyl lead additives is possible. Gasoline is acutely irritating to the eye, animal studies indicate no effect lasting longer than 7 days.

MacFarland (1982), reported on a chronic inhalation study of gasoline in Fischer 344 rats and $B6C3F_1$ mice. Exposure levels were 0, 67, 292 and 2056 ppm for 6 hours/day, 5 days/week for 103 to 113 weeks. Male (but not female) rats exhibited a progressive renal tubular disease and renal carcinomas in all dose groups; renal effects in mice were within the expected range of control. High dose female mice had an increased incidence of hepatocellular tumors (48%), but the spontaneous incidence of these tumors is also high (14%); males showed no increase (44% high dose vs. control 45%).

Reproductive Toxicity

Male rats exposed intermittently to about 650 ppm unleaded petrol for 2 months showed endocrine changes which were attributed to stress. The offspring of pregnant females exposed to 0, 400, and 1,600 ppm unleaded gasoline for 6 hours per day on days 6-15 of gestation did not show any teratogenic or fetotoxic effects. Mental retardation has been reported among the offspring of gasoline-sniffing mothers.

Genotoxicity

Negative results were observed with several common fuels when tested in the Ames Salmonella typhimurium assay, mouse lymphoma, and the rat bone marrow chromosomal aberration assay (Lebowitz et al. 1979). Unleaded gasoline did not induce unscheduled DNA synthesis in the male rat kidney at doses known to be nephrotoxic.

Carcinogenicity

As indicated earlier, chronic gasoline exposures produces renal tumors in rats.

REGULATIONS AND STANDARDS

The American Conference of Governmental Industrial Hygienists (ACGIH 1990) adopted a threshold limit value (TLV) of 300 ppm (mg/m^3) for gasoline vapors. Because of the complexity and variability in composition, OSHA has no standard but regulates the toxic components by their respective PELs (i.e., n-hexane, benzene, alkyl lead).

Gasoline as such is not mentioned in HEAST (1990) as having a specific cancer slope factor (CSF) or reference dose (RfD). However, individual components such as benzene, other aromatics, and n-hexane that have CSF or RfD values should be evaluated individually.

COMPOUND CAS NO.	ACGIH TLV ppm	RfD (inhal) mg/kg/day	RfD (oral) mg/kg/day	SLOPE FACTOR (inhale) mg/kg/day	SLOPE FACTOR (oral) mg/kg/day
Benzene 71-43-2	0.1	N/A	N/A	2.9E-2	2.9E-2
Ethylene dibromide 106-93-4	A2[1]	N/A	N/A	7.6E-1	8.5E+1
Ethylene dichloride 107-06-2	10	N/A	N/A	9.1E-2	9.1E-2
n-Hexane 110-54-3	50	6E-1	2E-1	N/A	N/A
Tetraethyl lead 78-00-2	0.1[2]	1E-7	2.9E-8	N/A	N/A

1 A2 - Substance classed as a suspected human carcinogen, no ACGIH TLV listed.
2 mg/m^3, not ppm.

REFERENCES

ACGIH. 1990. Threshold Limit Values for Chemical Substances and Biological Exposure Indices for 1990-1991. Cincinnati, Ohio: American Conference of Governmental Industrial Hygienists.

Donaldson, S.G. 1990. Volatilization of Gasoline from Contaminated Soil. M.S. Thesis, University of Reno.

Hanis, N.M., T.M. Holmes, L.G. Shallenberger, and K.E. Jones. 1982. Epidemiologic Study of Refinery and Chemical Plant Workers. J. Occup. Med. 24:203-212.

HEAST. 1990. Health Effects Assessment Summary Tables, Environmental Protection Agency. Office of Emergency and Remedial Response. EPA, OERR 9200 6-303, (90-4).

Kaplan, S. 1986. Update of a Mortality Study of Workers in Petroleum Refineries. J. Occup. Med. 28:514-516.

Korte, F. and W. Klein. 1982. Degradation of Benzene in the Environment. Ecotox. Environ. Safety 6:311-327.

Lee, T. and W. Seymour. 1979. Pneumonitis Caused by Petrol Siphoning. Lancet 8134:149.

Lebowitz, H., D. Brusick, and D. Matheson et al. 1979. Commonly Used Fuels and Solvents Evaluated in a Battery of Short-term Bioassays. Environ. Mutagen. 1:172-173.

MacFarland, H.N. 1982. Chronic Gasoline Toxicology. In: Proceedings of the Symposium: The Toxicology of Petroleum Hydrocarbons, American Petroleum Institute, Washington, DC.

McDermott, H. and S. Killiany. 1978. Quest for a Gasoline TLV. Am. Ind. Hyg. Assoc. J. 39:110-117.

Song, H.G. 1988. Petroleum Hydrocarbons in Soil: Biodegradation and Effects on the Microbial Community. Ph.D. Thesis, University of Medicine and Dentistry of New Jersey.

Thomas, T.L., R.J. Waxweiler, and R. Moure-Eraso et al. 1982. Mortality Patterns Among Workers in Three Texas Oil Refineries. J. Occup. Med. 24:135-141.

Wong, O. 1987. An Industry Wide Mortality Study of Chemical Workers Occupationally Exposed to Benzene. Br. J. Ind. Med. 44:365-381.

Von Burg, R. 1989. Toxicology Update for Gasoline. J. Appl. Toxicol. 9:203-210.

TOXICOLOGY PROFILE FOR WASTE OIL (RRPH)

GENERAL DATA

"Waste Oil" is a generic term commonly used to describe a range of petroleum compounds from heavy fuels to grease.

As much as one to two percent of the world's crude oil is refined to produce lubricating oil (24 million tons) (Vazquez-Duhalt 1989). The composition of waste oil is extremely variable and depends upon the original crude oil source, type of processing and refining, blending, additives, and use history. Waste oil may therefore range from virgin product accidentally spilled to used machine or automotive oil.

Petroleum oils are produced from the middle to heavy distillate fractions of crude oil. Because of the high boiling points for these fractions, the aromatic hydrocarbons benzene, ethylbenzene, toluene, and xylenes, typically found in lighter fuels will not be present in oils in high concentrations. These fractions may be further processed or treated to remove unwanted materials such as nitrogen, sulfur, metals, or polynuclear aromatic hydrocarbons (PAHs). For the most part, oils destined for the consumer market have been laundered to a very low content of PAHs. However, oils in refinery spills may contain several hundred ppm of PAHs. Used motor oil contains Pb, Zn, Cu, Cd, Cr, Ni, and other metals. Lead is the most abundant metal in motor oil and may be present at a concentration high as one percent (Vazquez-Duhalt 1989).

FATE AND TRANSPORT

Oil with characteristics (e.g. vapor pressure, viscosity) closer to fuel oils may volatilize to some extent to the air. However, microbial degradation will, more than likely, be the primary mechanism for the mineralization of spilt material. As much as 90 percent of the material resembling jet fuel may be removed by a combination of evaporation and microbial degradation with a half-life of one to eight weeks; in contrast, heavier fractions that resemble bunker oil (C_{15} and above) may be degraded only 25 to 30 percent and may be extremely persistent in soils (Song 1988). Given the correct circumstances, waste oils can percolate through the soil and float on the ground water. When spilled onto surface water, waste oils can be toxic to fish, waterfowl, and algae, but this is highly dependent upon the characteristics of the oily material and the size of the spill.

Approximately 30 percent of waste motor oil and lubricants produced are released into the environment. Because of the large quantities involved, the persistence of oil residues in the environment, and the potential for ecotoxicity, waste oils are an important environmental concern (Vazquez-Duhalt 1989).

When oil is spilt onto soil, it fills the spaces between the soil particles and hampers oxygen access, thereby promoting anaerobic zones. On the periphery of these oil-soiled zones, aerobic bacteria are promoted. Hence, these outer zones show increased nitrifying, denitrifying, ammonifying, and hydrocarbon-oxidizing microorganisms. The activity of these organisms in the outer zones increases the concentration of easily accessible substrates which stimulates an

increase in the numbers of anaerobic nitrogen fixing bacteria (Vazquez-Duhalt 1989). Thus, under certain circumstances, oil addition to soil can function as an amendment and increase the productivity of the soil.

TOXICITY DATA

Human Toxicology

In general, most oily materials derived from petroleum have a low order of toxicity. Inhalation of components of waste oil at concentrations sufficient to achieve a level of intoxication is not likely at normal temperatures and pressures. An attempt to generate a kerosene (diesel) laden atmosphere only resulted in an ambient concentration of 14 ppm (Carpenter et al. 1976). However, under certain occupational settings like tank cleaning, it may be possible to generate mists or aerosols that can lead to symptoms of overexposure. These symptoms may include headache, dizziness, nausea, gastrointestinal symptoms, shortness of breath, weakness, confusion, drowsiness, and possibly death (HSDB 1991). A single report considered chronic repeated exposure to an oil mist for 17 years to be the cause of lipid pneumonia in workers "heavily" exposed (Proctor et al. 1989).

Ingestion of petroleum waste oil (other than fuel oil) either accidentally, intentionally, or from contaminated well-water is not expected to have a significant effect except possibly induction of gastrointestinal hypermotility and diarrhea (MacFarland et al. 1982), and nausea and vomiting. Ingestion of the heavier waste oils is not expected to be complicated by aspiration into the lung, which produces a potentially lethal hemorrhagic pneumonitis (Lee and Seymour 1979).

Mammalian Toxicology and Significant Studies

The acute oral and dermal LD_{50} of petroleum waste oil is expected to be greater than 5 g/kg, or practically non toxic. Diesel fuel has an acute oral LD50 is in the range of 9 ml/kg body weight. New or used motor oil has an LD50 of 25 ml/kg, as does heavy fuel #6. Other properties, such as eye irritation, have ratings of practically non-irritating to mildly irritating. Skin irritation scores are similarly low ranging from non-irritating to mildly irritating (Beck et al. 1982). There are no readily available reports on hypersensitivity responses to waste oils, but sensitization is an expected effect because refined products in this distillation range have been reported to produce hypersensitivity reactions (Beck et al. 1982). Dermal absorption of oil can also be expected but the oil itself is unlikely to be the cause of systemic toxicity. Any toxicity is more likely to be attributable to a concomitant absorption of some oil contaminant.

Exposure of CD-1 mice to diesel vapor for eight hours per day on five consecutive days resulted in a decrement of performance on the roto-rod test, square box activity test, and hot plate test. However, the corneal reflex and inclined plane test was unaffected. General observations included vasodilation, ataxia, poor grooming, and in some cases tremor (Kainz and White 1982).

Exposure of rats to aerosolized diesel fuel at concentrations up to 6 mg/L produced direct toxic effects on the lungs but did not produce any neurotoxicity (Dalbey et al. 1987).

Reproductive Toxicity

Female rats were exposed 6 hours per day to air concentrations of 0, 100, and 400 ppm during days 6 through 15 of gestation. Neither jet fuel or No. 2 Fuel Oil produced any significant detrimental effects on the reproductive parameters of the experimental animals (Beliles and Mecler 1982). Neither Jet Fuel A or diesel fuel at exposure levels of 400 ppm, six hours per day, five days per week, for eight weeks reduced the fertility of CD-1 male mice (API 1980a,b).

External application of new or used motor oil to the egg shell of a number of bird species caused embryotoxicity and lethality. The used motor oil was more toxic than the new motor oil (Hoffman et al. 1982).

Genotoxicity

Ames testing of several common fuel oils produced mainly negative results. However, the "Modified Ames Assay", introduced by Blackburn et al. (1988), did demonstrate mutagenicity in two straight run gas oils that were previously considered to be negative. Diesel fuel was negative in a mouse lymphoma assay but positive on the rat bone marrow cytogenetics assay when administered by intraperitoneal injection (Conaway et al. 1982). Heating oil #2 did produce a positive Ames test and positive results in two other short term bioassays (Rothman and Emmett 1988).

Used motor oil has been shown to be highly mutagenic to Salmonella bacteria (Peake and Parker 1980). New crankcase motor oil initially tested negative with the standard Ames Assay but after an extraction procedure to remove "interfering chemicals", a dose dependent mutagenic response was observed with both gasoline and diesel crankcase oils. The extracts of the new motor oils, however, are considerably less mutagenic than the Used Crankcase Oil extracts. This effect can be explained by the fact that, during engine operation, the oil accumulates combustion dust and PAH formed in the combustion process or directly from the fuel (Thony et al. 1975). Extracts from the diesel and gasoline type engines were about equally potent (Dutcher et al. 1986).

Carcinogenicity

In classical mouse skin-painting bioassays, all petroleum fractions derived from a crude oil source that boiled between 120°F and 700°F showed a low level of tumorigenic activity (Lewis et al. 1982). Home heating oil also showed a low degree of tumorigenicity in a more recent mouse skin-painting assay (Witschi et al. 1987). Topical application of used motor oil from gasoline driven vehicles increased the incidence of local tumors in a dose related fashion. The application of new motor oil to mouse skin did not induce skin tumors (Saffiotti and Shubik 1963). This information plus the demonstrated mutagenic potential of used motor oils and their PAH content, allows a determination that such oils can be considered to be potentially carcinogenic (IARC 1984).

In a case referent study, Seimiatycki et al. (1987) reported an increase of several specific cancers associated with exposures to different petroleum products. Leaded gasoline was associated with stomach cancer; aviation gasoline with kidney cancer; diesel fuel with non adenocarcinoma of

the lung and prostate cancer; and mineral spirits with squamous cell lung cancer. However, not all parameters of concern were properly controlled, excluded, or assessed, making conclusions from this study unreliable.

IARC (1989) has classified gasoline, diesel fuel, and residual oil Category 2B, having limited evidence of carcinogenicity in animals and inadequate evidence in humans. Used motor oil (crankcase oil) is also classified as a category 2B. Light fuel oils, crude oil, and jet fuels have been classified as Category 3, having inadequate evidence of carcinogenicity in either animals or humans.

REGULATIONS AND STANDARDS

Neither the American Conference of Governmental Industrial Hygienists (ACGIH) nor OSHA have recommended or established permissible exposure standards (PELs) for diesel fuels or waste oils. NIOSH has recommended a 10 hour TWA of 100 mg/m^3 for kerosene or 14 ppm (MMWR. 37:24). The ACGIH (1991) and OSHA (1985) recommend a TLV of 5 mg/m^3 for Oil Mists.

Diesel fuels are not mentioned in HEAST (1990), nor identified for a specific cancer Slope Factor (CSF) or reference dose (RfD). However, individual components such as benzene, other aromatics, and n-hexane that have CSF or RfD values should be evaluated individually.

REFERENCES

API. 1980a. Mutagenicity Evaluation of Jet Fuel A in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-03. American Petroleum Institute Medical Research Publications, Washington, D.C.

API. 1980b. Mutagenicity Evaluation of Diesel Fuel in the Mouse Dominant Lethal Assay. Final Report. Litton Bionetics Inc. Proj. No. 21141-04. American Petroleum Institute Medical Research Publications, Washington, D.C.

Beck, L.S., D.I. Hepler, and K.L. Hansen. 1982. The Acute Toxicology of Selected Petroleum Hydrocarbons. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.

Beliles, R.P. and F.J. Mecler. 1982. Inhalation Teratology of Jet Fuel, Fuel Oil and Petroleum Naphtha in Rats. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.

Blackburn, G.R., R.A. Deitch, and T.A. Roy et al. 1988. Estimation of the Dermal Carcinogenic Potency of Petroleum Fractions Using a Modified Ames Assay. In: Polynuclear Aromatic Hydrocarbons: A Decade of Progress, Proceedings of the Tenth International Symposium. M. Cooke and A.J. Dennis, editors. Battelle Press, Columbus, Ohio. pp. 83-97.

Carpenter, C.P., D.L. Geary, and R.C. Meyers et al. 1976. Petroleum Hydrocarbon Toxicity Studies XI. Animal and Human Response to Vapors of Deodorized Kerosene. Toxicol. Appl. Pharmacol. 36:443-456.

Clayton, G.D. and F.E. Clayton. 1982. Patty's Industrial Hygiene and Toxicology. 3rd Edition. G.D. Clayton and F.E. Clayton, editors. John Wiley and Sons, Publishers, New York, New York.

Conaway, C.C., C.A. Schreiner, and S.T. Cragg. 1982. Mutagenicity Evaluation of Petroleum Hydrocarbons. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.

Dalbey, W., M. Henry, and R. Holmberg et al. 1987. Role of Exposure Parameters in the Toxicity of Aerosolized Diesel Fuel in the Rat. J. Appl. Toxicol. 7:265-275.

Dutcher, S.J., A.P. Li, and R.O. McClellan. 1986. Mutagenicity of Used Crankcase Oils from Diesel and Spark Ignition Automobiles. Environ. Res. 40:155-163.

HEAST. 1990. Health Effects Assessment Summary Tables, EPA, OERR 9200 6-303, (90-4).

Hoffman, D.J., W.C. Eastin, and M.L. Gay. 1982. Embryotoxic and Biochemical Effects of Waste Crankcase Oil on Bird Eggs. Toxicol. Appl Pharmacol. 63:230-241.

HSDB. 1990. Hazardous Substances Data Bank, National Library of Medicine. Washington, D.C.

IARC. 1984. International Agency for Research on Cancer. Vol 33. Polynuclear Aromatic Hydrocarbons: Part 2. Carbon Black, Mineral Oils and Some Nitroarenes. World Health Organization, Lyon, France.

IARC. 1989. International Agency for Research on Cancer. Vol 45. Occupational Exposures in Petroleum Refining; Crude Oil and Major Petroleum Fuels. World Health Organization, Lyon, France.

Kainz, R.J. and L.E. White. 1982. Consequences Associated with the Inhalation of Uncombusted Diesel Vapor. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.

Kirk Othmer. 1984. Diesel Fuel. In: Kirk-Othmer Encyclopedia of Chemical Technology. F. Mark, M. Grayson, and D. Eckroth et al., editors. 3rd Edition. John Wiley and Sons, New York.

Kryzanovskij, N.V. 1971. Occupational Health Conditions Associated with the Cleaning of Oil Tankers and their Effect on Worker's Health. Gig. Tr. Prof. Zabol. 15:14-17.

Lee T. and W. Seymour. 1979. Pneumonitis Caused by Petrol Siphoning. The Lancet 8134:149.

Lebowitz H., D. Brusick, and D. Matheson et al. 1979. Commonly Used Fuels and Solvents Evaluated in a Battery of Short-term Bioassays. Environ. Mutagen. 1:172-173.

Lewis, S.C., R.W. King, S.T. Cragg, and D.W. Hillman. 1982. Skin Carcinogenic Potential of Petroleum Hydrocarbons. 2. Carcinogenesis of Crude Oil, Distillate Fractions and

Chemical Class Subfractions. In: Proceedings of the Symposium on The Toxicology of Petroleum Hydrocarbons. H.N. MacFarland et al. (editors), American Petroleum Institute, Publisher. Washington, D.C. May.

NIOSH. 1977. Criteria for a Recommended Standard...Occupational Exposure to Refined Petroleum Solvents. National Institute for Occupational Safety and Health, Cincinnati, Ohio. NIOSH Publication #77-192.

OHM TADS. 1991. Oil and Hazardous Materials - Technical Assistance Data Systems. Environmental Protection Agency, Emergency Response Division, Washington D.C.

Peake, E. and K. Parker. 1980. Polynuclear Aromatic Hydrocarbons and the Mutagenicity of Used Crankcase Oil. In: A. Bjorseth and J. Dennis, editors. Polynuclear Aromatic Hydrocarbons: Chemistry and Biological Effects. Battelle Press, Columbus, Ohio. pp 1025-1039.

Rothman, N. and E.A. Emmett. 1988. The Carcinogenic Potential of Spetroleum-derived Products. Chapter 7. In: Occupational Medicine: State of the Art Reviews. 3:475-482.

Saffiotti, U. and P. Shubik. 1963. Studies on Promoting Action in Skin Carcinogenesis. National Cancer Inst. Monogr. 10:489-507.

Siemiatycki, J., R. Dewar, and L. Nadon et al. 1987. Associations Between Several Sites of Cancer and Twelve Petroleum Derived Liquids. Scan. J. Work Environ. Health. 13:493-504.

Song, H.G. 1988. Ph.D. Thesis, University of Medicine and Dentistry of New Jersey. Petroleum Hydrocarbons in Soil: Biodegradation and Effects on the Microbial Community.

Thony, C., J. Thony, and M. Lafontaine et al. 1975. Concentration En Hydrocarbons Polycycliques Aromatiques Concerogenes Del Ques Huiles Minerales. Arch. Mal. Prof. Med. Travail Sec. Soc. 36: 281-300.

Vazquez-Duhalt, R. 1989. Environmental Impact of Used Motor Oil. Sci. Total Environ. 79:1-23.

Witschi, H.P., L.H. Smith, and E.L. Frome et al. 1987. Skin Tumorigenic Potential of Crude and Refined Coal Liquids and Analogous Petroleum Products. Fund. Appl. Toxicol. 9:297-303.

BIS(2-ETHYLHEXYL)PHTHALATE

Bis(2-ethylhexyl)phthalate, also known as di-ethylhexyl phthalate (DEHP), is readily absorbed following oral or inhalation exposure (EPA 1980). Acute oral exposure (600 - 2,000 mg/kg/day DEHP) to rodents, guinea pigs, and monkeys has resulted in adverse effects on the livers and kidney (weight changes and enzyme induction) (ATSDR 1991). Chronic exposure to relatively high concentrations (200 mg/kg/day) of DEHP in the diet can cause retardation of growth and increased liver and kidney weights in laboratory animals (NTP 1982, EPA 1980, Carpenter et al. 1953). Effects on the liver at the cellular level were noted in rats at doses as low as 10 to 50 mg/kg/day DEHP (Ganning et al. 1989, Mitchell et al. 1985, Short et al. 1987) but dogs receiving doses of 59 mg/kg/day DEHP for one year had no observed changes in liver weight or structure (Carpenter et al. 1953, ATSDR 1991). Single oral doses of 4,882 and 9,756 mg/kg DEHP administered to pregnant rats on day 12 of gestation caused a dose-related increase in dead and resorbed fetuses and a number of malformations in the survivors (Ritter et al. 1989). DEHP is lipophilic and has the potential to be transported in maternal milk and thus have an impact on postnatal development (ATSDR 1991). Studies in rodents exposed to doses in the range of 200 to 2,800 mg/kg/day DEHP indicate that the testes are a primary target tissue resulting in increased testicular weights, tubular atrophy, decreased male fertility, and abnormal sperm (ATSDR 1991). Several chronic feeding studies in rodents indicate that lifetime exposure to 300 to 1,000 mg/kg/day DEHP can cause liver tumors in rats and mice (Kluwe et al. 1982, Rao et al. 1987, 1990).

EPA (1993a) classified DEHP in Group B2--Probable Human Carcinogen. EPA (1993) calculated an oral cancer slope factor for DEHP of 1.4×10^{-2} $(mg/kg/day)^{-1}$ based on data from the NTP (1982) study in which liver tumors were noted in mice. EPA recommended an oral reference dose (RfD) for DEHP of 2×10^{-2} mg/kg/day for both chronic (EPA 1993a) and subchronic (EPA 1993b) exposures based on a study by Carpenter et al. (1953) in which increased liver weight was observed in female guinea pigs exposed to 19 mg/kg bw/day in the diet for 1 year; an uncertainty factor of 1,000 was used to develop both RfDs.

REFERENCES

Agency for Toxic Substance and Disease Registry (ATSDR). 1991. Toxicological Profile for Di(2-ethylhexyl)phthalate. Draft. U.S. Department of Health and Human Services. October.

Carpenter, C.P., Weil, C.S., and Smyth, H.F. 1953. Chronic oral toxicity of di(2-ethylhexyl)phthalate for rats, guinea pigs, and dogs. Arch. Indust. Hyg. Occup. Med. 8:219-226.

Environmental Protection Agency (EPA). 1980. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 40/5-80-067.

Environmental Protection Agency (EPA). 1993a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

Environmental Protection Agency (EPA). 1993b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment. Environmental Assessment and Criteria Office, Cincinnati, Ohio for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. FY-1993.

Ganning, A.E., Olsson, M.J., Peterson, E. et al. 1989. Fatty acid oxidation in hepatic peroxisomes and mitochondria after treatment of rats with di(2-ethylhexyl)phthalate. Pharmacol Toxicol 65:265-268 (as cited in ATSDR 1991).

Kluwe, W.M., Haseman, J.K., Douglas, J.F. et al. 1982. The carcinogenicity of dietary di(2-ethylhexyl)phthalate in Fischer 344 rats and B6C3F1 mice. J Toxicol Environ Health 12:159-169.

Mitchell, F.E., Price, S.C., Hinton, R.H. et al. 1985. Time and dose-response study of the effects on rats of the plasticizer di(2-ethylhexyl)phthalate. Toxicol Appl Pharmacol. 81:371-392 (as cited in ATSDR 1991).

National Toxicology Program (NTP). 1982. Carcinogenesis Bioassay of Di(2-ethylhexyl)phthalate in F344 Rats and $B6C3F_1$ Mice. Feed Study. NTP Technical Report Series No. 217, U.S. Department of Health and Human Services. NIH Publication No. 82-1773. NTP-80-37.

Rao, M.S., Usuda, N., Subbarao, V. et al. 1987. Absence of gamma-glutamyl transpeptidase activity in neoplastic lesions induced in the liver of male F-344 rats by di(2-ethylhexyl)phthalate, a peroxisome proliferator. Carcinogenesis 8:1347-1350.

Rao, M.S., Yeldandi, A.V., Subbarao, V. 1990. Quantitative analysis of hepatocellular lesions induced by di(2-ethylhexyl)phthalate in F-344 rats. J Toxicol Environ Health 30:85-89.

Ritter, E.J., Scott, W.J. Jr., Randall, J.L. et al. 1987. Teratogenicity of Di(2-ethylhexyl)phthalate, 2-ethylhexanol, 2-ethylhexanoic acid, and valpoic acid, and potentiation by caffeine. Teratology 35:41-46.

Short, R.D., Robinson, E.C., Lington, A.W. et al. 1987. Metabolic and peroxisome proliferation studies with di(2-ethylhexyl)phthalate in rats and monkeys. Toxicol Ind Health 3:185-195 (as cited in ATSDR 1991).

1,2-DICHLOROETHANE

1,2-Dichloroethane (1,2-DCA) is lipophilic and is absorbed through oral, inhalation and dermal exposure. Data from animal studies suggest that absorption is rapid and complete with at least 90 percent of inhaled or orally administered dose absorbed (Reitz et al. 1980). Effects of acute inhalation exposure in humans include CNS effects such as nausea, irritability, headache, drowsiness and tremors, liver and kidney effects and respiratory distress (ATSDR 1992). Acute oral exposure in humans resulted in adverse liver, kidney and CNS effects. Evidence from animal studies suggests that the immune system is a target of 1,2-DCA toxicity following acute oral exposure. Subchronic exposure of rats given gavage doses of greater than or equal to 240 mg/kg/day resulted in mild hyperplasia and inflammation of the forestomach (NTP 1991). In the same study, neurological effects were manifested as tremors, salivation, emaciation, abnormal posture, ruffled fur and dyspnea (NTP 1991). Chronic inhalation studies in animals also have revealed toxic effects including degeneration of the liver (EPA 1985). Overall evidence suggests that 1,2-DCA is not a developmental toxicant in experimental animals except at maternally toxic levels (ATSDR 1992; EPA 1985), however intermittent exposure of females to 4.7 ppm for 4 months prior to mating followed by inhalation exposure during pregnancy produced a statistically significant increase in embryo mortality (Vozovaya 1977). Additionally, nursing women exposed to 1,2-DCA in the workplace air accumulate the chemical in breast milk (Erikson et al. 1980; Urosova 1953). In long-term oral bioassays sponsored by the National Cancer Institute (NCI 1978), increased incidence of squamous-cell carcinomas of the forestomach, mammary gland adenocarcinomas, and hemangiosarcomas have been observed in rats exposed to 1,2-DCA; pulmonary adenomas, mammary adenocarcinomas, and uterine endometrial tumors have been observed in mice exposed to this chemical.

EPA (1993) has classified 1,2-DCA in Group B2 (Probable Human Carcinogen) based on inadequate evidence of carcinogenicity from human studies and sufficient evidence of carcinogenicity from animal studies. EPA (1993) derived an oral cancer slope factor of 9.1×10^{-2} $(mg/kg/day)^{-1}$ for 1,2-DCA based on the incidence of hemangiosarcomas in Osborne-Mendel male rats observed in the NCI (1978) gavage study. An inhalation cancer unit risk of 2.6×10^{-5} $(\mu g/m^3)^{-1}$ has also been calculated by EPA (1993) using the same gavage study, and route-to-route extrapolation.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological Profile for 1,2-Dichloroethane. U.S. Department of Health and Human Services. Draft for Public Comment. October.

Environmental Protection Agency (EPA). 1984. Health Effects Assessment for 1,2-Dichloroethane. Environmental Criteria and Assessment Office, Cincinnati, Ohio. September 1984. EPA 540/1-86-002.

Environmental Protection Agency (EPA). 1985. Health Assessment Document for 1,2-Dichloroethane. Office of Health and Environmental Assessment, Washington, D.C. September 1985. EPA 600/8-84-006F.

Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

Erickson, M.D., B.S.H. Harris, III., and E.D. Pellizzari et al. 1980. Acquisition and Chemical Analysis of Mother's Milk for Selected Toxic Substances. Report to U.S. EPA, Office of Pesticides and Toxic Substances, Washington, D.C., by Research Triangle Park Institute, RTP, North Carolina. EPA 560/13-80/029 (as cited in ATSDR 1992).

National Cancer Institute (NCI). 1978. Bioassay of 1,2-Dichloroethane for Possible Carcinogenicity. NCI Carcinogenesis Technical Report Series No. 55. Washington, D.C. DHEW (NIH) Publication No. 78-1361.

National Toxicology Program (NTP). 1991. NTP Report on the Toxicity of 1,2-Dichloroethane in F344/N Rats, Sprague-Dawley Rats, Osborne-Mendel Rats and B6C3F Mice (Drinking Water and Gavage Studies). NTP, U.S. Department of Health and Human Services, Public Health Service, National Institute of Health, Research Triangle Park, North Carolina (as cited in ATSDR 1992).

Reitz, R.H., T.R. Fox, and J.Y. Domoradzki et al. 1980. Pharmacokinetics and Macromolecular Interactions or Ethylene Dichloride: Comparison of Oral and Inhalation Exposures. In: B.N. Ames, P. Infante, and R. Reitz, Eds. Ethylene Dichloride: A Potential Health Risk? Cold Spring Harbor, New York: Cold Spring Harbor Laboratory, 135-148 (as cited in ATSDR 1992).

Urosova, T.P. 1953. [The Possible Presence of Dichloroethane in Human Milk With Exposure in Industrial Conditions.] Gig. Sanit. 18:36-37 (as cited in ATSDR 1992).

Vozovaya, M. 1977. [The Effect of Dichloroethane on the Sexual Cycle and Embryogenesis of Experimental Animals.] Akusk Ginekol (Moscow) 2:57-59 (as cited in ATSDR 1992).

TETRACHLOROETHENE

Tetrachloroethene is absorbed following inhalation (IARC 1979) and oral (EPA 1985a,b) exposure. Tetrachloroethene vapors and liquid also can be absorbed through the skin (EPA 1985a,b). The principal toxic effects of tetrachloroethene in humans and animals following acute and longer-term exposures include CNS depression and fatty infiltration of the liver and kidney with concomitant changes in serum enzyme activity levels indicative of tissue damage (EPA 1985a,b; Buben and O'Flaherty 1985). Humans exposed to doses of between 136 and 1,018 mg/m^3 for 5 weeks develop central nervous system effects, such as lassitude and signs of inebriation (Stewart et al. 1974). The offspring of female rats and mice exposed to high concentrations of tetrachloroethene for 7 hours daily on days 6-15 of gestation developed toxic effects, including a decrease in fetal body weight in mice, and a small but significant increase in fetal resorption in rats (Schwetz et al. 1975). Mice also exhibited developmental effects, including subcutaneous edema and delayed ossification of skull bones and sternebrae (Schwetz et al. 1975). In a National Cancer Institute bioassay (NCI 1977), an increased incidence of hepatocellular carcinoma was observed in both sexes of B6C3F1 mice administered tetrachloroethylene in corn oil by gavage for 78 weeks. Increased incidence of mononuclear cell leukemia and renal adenomas and carcinomas (combined) have been observed in long term bioassays in which rats were exposed to tetrachloroethene by inhalation (NTP 1986).

Tetrachloroethene is currently under review by the Carcinogen Risk Assessment Verification Endeavor (CRAVE) and estimates of cancer potency were recently withdrawn by EPA (1992b). However, the EPA Environmental Criteria and Assessment Office (ECAO) (1992a) currently classifies tetrachloroethene as a Group B2/C carcinogen (Probable/Possible Human Carcinogen). ECAO (1992a) has reported an oral slope factor of 5.2×10^{-2} (mg/kg/day)$^{-1}$ based on liver tumors observed in the NCI (1977) gavage bioassay for mice. An inhalation cancer unit risk of 5.8×10^{-7} (μg/m^3)$^{-1}$ is based on an NTP (1986) bioassay in rats and mice in which leukemia and liver tumors were observed (ECAO 1992a). Both the cancer slope factor and unit risk are currently under review by EPA. EPA (1993) also derived an oral RfD of 1×10^{-2} mg/kg/day for tetrachloroethene based on a six-week gavage study by Buben and O'Flaherty (1985). In this study, liver weight/body weight ratios were significantly increased in mice and rats treated with 71 mg/kg-day tetrachloroethene but not in animals treated with 14 mg/kg-day. The RfD was derived using a NOAEL of 14 mg/kg/day and applying an uncertainty factor of 1,000. EPA (1992b) established a subchronic oral RfD of 1×10^{-1} mg/kg/day, using an uncertainty factor of 100 based on the same study and effect of concern.

The American Conference of Governmental Industrial Hygienists (ACGIH) has set a Short-Term Exposure Level - Threshold Limit Value of 200 ppm (1,000 mg/m^3) for tetrachloroethene (ACGIH 1991). The STEL-TLV is defined as a 15-minute time-weighted average which should not be exceeded at any time during a work day. A health criterion for acute inhalation exposure to tetrachloroethene of 100 mg/m^3 can be derived from the STEL-TLV by combining it with a safety factor of 10 to account for the healthy worker effect which assumes that employed persons are healthier than the general population.

REFERENCES

American Conference of Governmental Industrial Hygienist (ACGIH). 1991. Threshold Limit Values and Biological Exposure Indices for 1990-1991. Cincinnati, Ohio.

Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the Role of Metabolism in the Hepatotoxicity of Trichloroethylene and Perchloroethylene: A Dose-effect Study. Toxicol. Appl. Pharmacol. 78:105-122.

Environmental Protection Agency (EPA). 1985a. Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Office of Health and Environmental Assessment, Washington, D.C. July 1985. EPA 600/8-82-005F.

Environmental Protection Agency (EPA). 1985b. Drinking Water Criteria Document for Tetrachloroethylene. Office of Drinking Water, Criteria and Standards Division, Washington, D.C. June 1985.

Environmental Criteria and Assessment Office (ECAO). 1992a. Written Correspondence from Joan Dollarhide, Chemical Mixtures Assessment Branch, U.S. Environmental Protection Agency. July 16, 1992.

Environmental Protection Agency (EPA). 1992b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Remedial Response, Washington, D.C. FY-1992.

Environmental Protection Agency (EPA). 1993. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

International Agency for Research on Cancer (IARC). 1979. IARC Monographs on the Evaluation of the Carcinogenic Risks of Chemicals to Humans. Vol. 20: Some Halogenated Hydrocarbons. World Health Organization, Lyon, France.

National Cancer Institute (NCI). 1977. Bioassay of Tetrachloroethylene for Possible Carcinogenicity. CAS No. 127-18-4. NCI Carcinogenesis Technical Report Series No. 13, Washington, D.C. DHEW (NIH) Publication No. 77-813.

National Toxicology Program (NTP). 1986. Toxicology and Carcinogenesis Studies of Tetrachloroethylene (Perchloroethylene) (CAS No. 127-18-4) in F344/N Rats and B6C3F1 Mice (Inhalation Studies). NTP Technical Report Series No. 311, Research Triangle Park, North Carolina. DHEW (NIH) Publication No. 86-2567.

Schwetz, B.A., B.K.J. Leong, and P.J. Gehring. 1975. The Effect of Maternally Inhaled Trichloroethylene, Perchloroethylene, Methyl Chloroform, and Methylene Chloride on Embryonal and Fetal Development in Mice and Rats. Toxicol. Appl. Pharmacol. 55:207-219.

Stewart, R.D., C.L. Hake, H.V. Forster, A.J. Lebrun, J.F. Peterson, and A. Wu. 1974. Tetrachloroethylene: Development of a Biologic Standard for the Industrial Worker by Breath Analysis. Medical College of Wisconsin, Milwaukee, Wisconsin. NIOSH-MCOW-ENUM-PCE-74-6.

CADMIUM

Gastrointestinal absorption of cadmium in humans ranges from 5-6 percent (EPA 1985a). Pulmonary absorption of cadmium in humans is reported to range from 10 to 50 percent (CDHS 1986). Cadmium bioaccumulates in humans, particularly in the kidney and liver (EPA 1985a,b). Chronic oral or inhalation exposure of humans to cadmium has been associated with renal dysfunction, itai-itai disease (bone damage), hypertension, anemia, endocrine alterations, and immunosuppression. Renal toxicity occurs in humans at a renal cortex concentration of cadmium of 200 µg/g (EPA 1985b). Epidemiological studies have demonstrated a strong association between inhalation exposure to cadmium and cancers of the lung, kidney, and prostate (EPA 1985b; Thun et al. 1985). In experimental animals, cadmium induces injection-site sarcomas and testicular tumors. When inhaled, cadmium chloride is a potent pulmonary carcinogen in rats. Cadmium is a well-documented animal teratogen (EPA 1985b).

EPA (1996) classified cadmium as a Group B1 agent (Probable Human Carcinogen) by inhalation. This classification applies to agents for which there is limited evidence of carcinogenicity in humans from epidemiologic studies. EPA (1996) derived an inhalation unit risk of 1.8×10^{-3} $(\mu g/m^3)^{-1}$ for cadmium based on epidemiologic studies in which respiratory tract tumors were observed (Thun et al. 1985; EPA 1985b). Using renal toxicity as an endpoint, and a safety factor of 10, EPA derived two separate oral reference doses (RfDs) (EPA 1996a). The RfD associated with oral exposure to drinking water is 5×10^{-4} mg/kg/day, and is based upon the lowest-observed-adverse-effect level (LOAEL) of 0.005 mg/kg in humans (EPA 1985a, Friberg et al. 1974). The RfD associated with exposure to cadmium in food is 1×10^{-3} mg/kg/day.

REFERENCES

California Department of Health Services (CDHS). 1986. Report to the Scientific Review Panel on Cadmium. Part B. Health Effects of Cadmium. Revised. Prepared by the Epidemiological Studies and Surveillance Section, Berkeley, California. September 19, 1986.

Environmental Protection Agency (EPA). 1980. Ambient Water Quality Criteria Document for Cadmium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, Ohio for the Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-80-025.

Environmental Protection Agency (EPA). 1985a. Drinking Water Criteria Document for Cadmium. Final Draft. Office of Drinking Water, Washington, D.C. April 1985. PB86-117934.

Environmental Protection Agency (EPA). 1985b. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium. Addendum to the Health Assessment Document for Cadmium (May 1981; EPA/600/8-81/023). Office of Health and Environmental Assessment, Washington, D.C. June 1985. EPA 600/8-83-025F.

Environmental Protection Agency (EPA). 1987. Health Advisory for Cadmium. Office of Drinking Water, Washington, D.C. March 31, 1987.

Environmental Protection Agency (EPA). 1996. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

Friberg, L., M. Piscator, G.F. Nordberg, and T. Kjellstrom. 1974. Cadmium in the Environment. 2nd Ed. CRC Press, Inc., Boca Raton, Florida.

Thun, M.J., T.M. Schnorr, A.B. Smith, W.E. Halperin, and B.A. Lemen. 1985. Mortality Among a Cohort of U.S. Cadmium Production Workers - An Update. JNCI 74:325-333.

MANGANESE

Manganese is considered to be an essential element and among the least toxic of the trace metals (NRC 1989). The oral absorption of dietary manganese ranges from 3 to 10 percent (EPA 1993). However, manganese is absorbed to a greater extent following inhalation exposures. The National Research Council has established a provisional recommended dietary allowance for adults of 2 to 5 mg/day (NRC 1989). The effects following acute exposure to manganese are unknown.

Chronic occupational exposure to manganese dust (0.02 - 2.6 mg/m^3) has been associated with respiratory symptoms and pneumonitis (Chandra et al. 1981, 1990). Higher levels have been associated with a condition known as manganism, a progressive neurological disease characterized by speech disturbances, tremors, and difficulties in walking. For example, male workers exposed to manganese dioxide, tetroxide, and various salts [time-weighted-average (TWA) of total airborne manganese dust ranged from 0.07-8.61 mg/m^3] experienced an increased incidence of psychomotor disturbances (e.g., reaction time, hand-eye coordination, and hand steadiness) (Roels et al. 1987). Other effects observed in humans occupationally exposed to manganese dust include hematological (Chandra et al. 1981; Flinn et al. 1941; Kesic and Hausler 1954), cardiovascular (Saric and Hrustic 1975), and reproductive effects (Cook et al. 1974; Emara et al 1971; Lauwerys et al 1985; Rodier 1955).

In adults, a safe intake of manganese from dietary sources ranges from 2-10 mg/day (10 mg/day = 0.14 mg/kg/day) (WHO 1973; NRC 1989; Schroeder et al. 1966). Individuals who chronically ingested drinking water from natural wells containing manganese concentrations of 1,600 to 2,300 ug/L (0.06 mg/kg/day), showed a statistically significant increase in minor neurologic effects (neurologic exam scores) (Kondakis et al. 1989). Higher concentrations in drinking water (0.8 mg/kg/day) have resulted in symptoms including lethargy, increased muscle tonus, tremor, and mental disturbances (Kawamura et al. 1941).

The apparent differences in manganese toxicity following dietary and drinking water exposures can be attributed to the greater bioavailability of manganese from water (EPA 1993). Chronic oral exposure of rats to manganese chloride can also result in central nervous system dysfunction (Leung et al. 1981; Lai et al. 1982). Chronic inhalation exposure of experimental animals (monkeys, rats, mice, hamsters) has resulted in respiratory effects, however, other studies have demonstrated that these effects may be immunological in origin (ATSDR 1992).

Manganese has not been reported to be teratogenic but it has been observed to cause depressed reproductive performance and reduced fertility in humans and experimental animals (EPA 1984a). Certain manganese compounds have been shown to be mutagenic in a variety of bacterial tests. Manganese chloride and potassium permanganate can cause chromosomal aberrations in mouse mammary carcinomal cells. Manganese was moderately effective in enhancing viral transformation of Syrian hamster embryo cells (EPA 1984a,b).

EPA (1993a) established a weight-of-evidence classification for manganese of D (not classifiable as to human carcinogenicity). EPA (1993a) derived two separate oral reference doses (RfD). The separate RfDs for food and water indicate a potentially higher bioavailability of manganese from

drinking water than from the diet. The RfD associated with oral exposure to drinking water is 5×10^{-3} mg/kg/day based on a no-observed-adverse-effect-level (NOAEL) of 5×10^{-3} mg/kg/day for humans (Kondakis et al. 1989). EPA (1993a) also derived an RfD of 1.4×10^{-1} mg/kg/day for manganese in food based on a NOAEL of 0.14 mg/kg/day (10 mg/day) in humans chronically exposed to dietary levels (WHO 1973; Schroeder et al. 1966; NRC 1989). The effect of concern was on the central nervous system, and an uncertainty factor of one was used to derive both RfDs. The chronic RfD in food was adopted as the subchronic RfD (EPA 1993b). EPA (1993a) derived a chronic inhalation reference concentration (RfC) of 4×10^{-4} mg/m^3 based upon an occupational study conducted by Roels et al. (1987) in which respiratory symptoms and psychomotor disturbances were observed. EPA (1993b) adopted the chronic RfC as the subchronic RfC. An uncertainty factor of 900 was used to derive both RfCs.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR). 1992. Toxicological Profile for Manganese. U.S. Department of Health and Human Services, Center for Disease Control.

Chandra, S.V., G.S. Shukla, R.S. Striavastava, H. Singh, and V.P. Gupta. 1981. An Exploratory Study of Manganese Exposure to Welders. Clin. Toxicol. 18:407-416.

Cook, D.G., S. Fahn, and K.A. Brait. 1974. Chronic Manganese Intoxication. Arch. Neurol. 30:59-64.

Emara, A.M., S.H. El-Ghawabi, O.I. Madkour, and G.H. El-Sarma. 1971. Chronic Manganese Poisoning in the Dry Battery Industry. Br. J. Ind. Med. 28:78-82.

Environmental Protection Agency (EPA). 1984a. Health Assessment Document for Manganese. Final Report. Environmental Criteria and Assessment Office, Environmental Protection Agency, Cincinnati, Ohio. August 1984. EPA 600/8-83-013F.

Environmental Protection Agency (EPA). 1984b. Health Effects Assessment for Manganese (and compounds). Environmental Criteria and Assessment Office, Washington, D.C. EPA 540/1-86-057.

Environmental Protection Agency (EPA). 1993a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio.

Environmental Protection Agency (EPA). 1993b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. FY-1993.

Flinn, R.H., P.A. Neal, and W.B. Fulton. 1941. Industrial Manganese Poisoning. J. Ind. Hyg. Toxicol. 23:374-387.

Iregren, A. 1990. Psychological Test Performance in Foundry Workers Exposed to Low Levels of Manganese. Neurotox. Teratol. 12:673-675.

Kawamura, R., H. Ikuta, and S. Fukuzumi et al. 1941. Intoxication by Manganese in Well Water. Kitasato Arch. Exp. Med. 18:145-149.

Kesic, B. and V. Hausler. 1954. Hematological Investigation on Workers Exposed to Manganese Dust. Arch. Ind. Hyg. Occup. Med. 10:336-343.

Kondakis, X.G., M. Makris, and M. Leotsinidis et al. 1989. Possible Health Effects of High Manganese Concentration in Drinking Water. Arch. Environ. Health 44:175-178.

Lai, J.C.K., T.K.C.Leung, and L. Lim. 1982. Activities of the Mitochondrial NAD-linked Isocitric Dehydrogenase in Different Regions of the Rat Brain. Changes in Aging and the Effect of Chronic Manganese Chloride Administration. Gerontology 28:81-85.

Lauwerys, R., H. Roels, and P. Genet et al. 1985. Fertility of Wale Workers Exposed to Mercury Vapor or to Manganese Dust: A Questionnaire Study. Am. J. Ind. Med. 7:171-176.

Leung, T.K.C., J.C.K. Lai, and L. Lim. 1981. The Regional Distribution of Monoamine Oxidase Activities Towards Different Substrates: Effects in Rat Brain of Chronic Administration of Manganese Chloride and of Aging. J. Neurochem. 36:2037-2043.

National Research Council (NRC). 1989. Recommended Dietary Allowances, 10th Ed. Food and Nutrition Board, National Research Council, National Academy Press, Washington, D.C. 230-235.

Rodier, J. 1955. Manganese Poisoning in Moroccan Miners. Br. J. Ind. Med. 12:21-35.

Roels, H., R. Lauwerys, and J.P. Buchet et al. 1987. Epidemiological Survey Among Workers Exposed to Manganese: Effects on Lung, Central Nervous System, and Some Biological Indices. Am. J. Ind. Med. 11:307-327.

Saric, M. and O. Hrustic. 1975. Exposure to Airborne Manganese and Arterial Blood Pressure. Environ. Res. 10:314-318.

Schroeder, H.A., D.D. Balassa, and I.H. Tipton. 1966. Essential Trace Metals in Man: Manganese, a Study in Homeostasis. J. Chron. Dis. 19:545-571.

WHO (World Health Organization). 1973. Trace Elements in Human Nutrition: Manganese. Report of a WHO Expert Committee. Technical Report Service, 532, WHO, Geneva, Switzerland. 34-36.

VANADIUM

Pentavalent vanadium compounds are generally considered to be more toxic than other valence states. Many incidents of short-term and long-term occupational exposures to vanadium, mainly vanadium pentoxide dust, have been reported. Inhalation causes respiratory tract irritation, coughing, wheezing, labored breathing, bronchitis, chest pains, eye and skin irritation, and discoloration of the tongue (NIOSH 1977; NAS 1974). Effects seen in experimental animals following chronic inhalation exposure include fatty degeneration of the liver and kidneys, hemorrhage, and bone marrow changes (Browning 1969). Rats administered 0.77 mg/kg/day (5 ppm) vanadium in their drinking water showed no adverse effects (Schroeder et al 1970).

EPA (1993a) derived a chronic and subchronic oral reference dose (RfD) of 7×10^{-3} mg/kg/day based on a chronic study in which rats received vanadium in their drinking water (Schroeder et al. 1970). A no-observed-adverse-effect level (NOAEL) of 0.77 mg/kg/day (5 ppm) and an uncertainty factor of 100 were used to develop the RfD. EPA (1993b) established an oral RfD for vanadium pentoxide of 9×10^{-3} mg/kg/day. This value is based on a chronic rat study in which a NOAEL of 0.89 mg vanadium pentoxide/kg/day was noted. The only reported effect was a decrease in the amount of cystine in the hair (Stokinger et al. 1953). An uncertainty factor of 100 was used to calculate the vanadium pentoxide RfD. EPA has not developed inhalation criteria for vanadium.

REFERENCES

Browning, E. 1969. Toxicity of Industrial Metal. 2nd Ed. Appleton-Century-Crofts, New York.

Environmental Protection Agency (EPA). 1993a. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. FY-1993.

Environmental Protection Agency (EPA). 1993b. Integrated Risk Information System (IRIS). Health Criteria and Assessment Office, Cincinnati, Ohio.

National Academy of Sciences (NAS). 1974. Vanadium. Committee on Biological Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council, Washington, D.C.

National Institute of Occupational Safety and Health (NIOSH). 1977. Criteria for a Recommended Standard--Occupational Exposure to Vanadium. DHEW (NIOSH) Publication No. 77-222.

Schroeder, J.A., M. Mitchner, and A.P. Nason. 1970. Zirconium, Niobium, Antium, Antimony, Vanadium, and Lead in Rats: Life Term Studies. J. Nutr. 100(1):59-68.

Stokinger, H.E., W.E. Wagner, J.T. Moutain, F.R. Stacksill, O.J. Dobrogorski, and R.G. Keenan. 1953. Unpublished Results. National Institute for Occupational Safety and Health, Division of Occupational Health, Cincinnati, Ohio.

ZINC

Zinc is absorbed in humans following oral exposure (approximately 20-30 percent) (ATSDR 1992), however insufficient data are available to evaluate absorption following inhalation exposure (EPA 1984). Zinc is an essential trace element that is necessary for normal health and metabolism and therefore is nontoxic in trace quantities (Hammond and Beliles 1980). The National Research Council (NRC) recommends a dietary allowance of 10-15 mg/day for adults (NRC 1989). Exposure to zinc at concentrations that exceed recommended levels, however, has been associated with a variety of adverse effects. In humans, acute inhalation exposure to relatively high levels of zinc has been associated with gastrointestinal disturbances, dermatitis, and metal fume fever, a condition characterized by chest pain, cough, and dyspnea, as well as impaired pulmonary function characterized by reduced lung volumes (ATSDR 1992). Eighteen healthy women given supplements of zinc gluconate (1 mg/kg/day) for 10 weeks developed slight alterations in blood chemistry (decreased enzyme levels) (Yadrick et al. 1989). Chronic oral exposure of humans to zinc (2 mg/kg/day) may cause decreased red blood cell count (Hale et al. 1988). Experimental animals (rats, rabbits, mice) administered zinc in the diet (68 - 1,110 mg/kg/day) for durations up to 1 year manifested blood, liver, renal, and reproductive effects (ATSDR 1992). An increased incidence of fetal resorption was noted in pregnant rats administered 200 mg/kg/day zinc (Schlicker and Cox 1968). In addition, increased pre-implantation loss was observed in rats fed the same concentration for 18 days (Pal and Pal 1987). There is no evidence that zinc is carcinogenic (ATSDR 1992).

EPA (1993a) derived an oral reference dose (RfD) of 3×10^{-1} mg/kg/day based on a human diet supplement study in which decreased blood enzyme activity [47 percent decrease in erythrocyte superoxide dismutase (ESOD)] was observed in adult females after 10 weeks of zinc exposure of 1 mg/kg-day (59.72 mg/day) (Yadrick et al. 1989). An uncertainty factor of 3 was applied to the lowest-adverse-effect-level (LOAEL) of 59.72 mg/day (1 mg/kg/day) to derive the RfD. EPA (1993b) adopted the chronic RfD as the subchronic RfD.

REFERENCES

Agency for Toxic Substances and Disease Control (ATSDR). 1992. Toxicological Profile for Zinc. U.S. Department of Health and Human Services, Public Health Service. Draft for Public Comment. October.

Environmental Protection Agency (EPA). 1984. Health Effects Assessment for Zinc (and Compounds). Office of Emergency and Remedial Response, Washington, D.C. EPA 540/1-86-048. September 1984.

Environmental Protection Agency (EPA). 1993a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office. Cincinnati, Ohio.

Environmental Protection Agency (EPA). 1993b. Health Effects Assessment Summary Tables. Prepared by Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Cincinnati, Ohio, for the Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, D.C. FY-1993.

Hale, W.E., F.E. May, and R.G. Thomas et al. 1988. Effect of Zinc Supplementation on the Development of Cardiovascular Disease in the Elderly. J. Nutr. Elder 8(2):49-57.

Hammond, P.B. and R.P. Beliles. 1980. Metals. In J. Doull, C.D. Klaassen, and M.O. Amdur, Eds. Casarett and Doull's Toxicology: The Basic Science of Poisons. 2nd Ed. Macmillan Publishing Co., New York. Pp. 409-467.

National Research Council (NRC). 1989. Recommended Dietary Allowances, 10th Ed. Food and Nutrition Board, National Research Council, National Academy Press, Washington, D.C. 230-235.

Pal, N. and B. Pal. 1987. Zinc Feeding and Conception in the Rat. Int. J. Vitamin Nutr. Res. 57:437-440.

Schlicker, S.A. and P.H. Cox. 1968. Maternal Dietary Zinc, and Development and Zinc, Iron, and Copper Content of the Rat Fetus. J. Nutr. 95:287-294.

Yadrick, M.K., M.S. Kenney, and E.A. Winterfelt. 1989. Iron, Copper and Zinc Status: Response to Supplementation with Zinc or Zinc and Iron in Adult Females. Am. J. Clin. Nutr. 49:145-150.

APPENDIX C

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS
CF = CS*Bv*%V

SPECIES brown lemming COC	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
Inorganics				
Aluminum	0	0.004	1.00	0.000
Cadmium	19	0.550	1.00	10.450
Iron	57,000	0.004	1.00	228.000
Lead	30	0.045	1.00	1.350
Manganese	610	0.250	1.00	152.500
Zinc	81	1.500	1.00	121.500
Organics				
DRPH	3,190	0.033	1.00	106.756
Ethylbenzene	0.32	0.585	1.00	0.187
Xylenes (total)	1	0.970	1.00	0.970
Tetrachloroethene	1.1	1.336	1.00	1.469
Naphthalene	0.1	0.443	1.00	0.044
Trimethylbenzene	1.4	0.253	1.00	0.354

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS
CF = CS*Bv*%V

SPECIES caribou COC	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
Inorganics				
Aluminum	0	0.004	1.00	0.000
Cadmium	19	0.550	1.00	10.450
Iron	57,000	0.004	1.00	228.000
Lead	30	0.045	1.00	1.350
Manganese	610	0.250	1.00	152.500
Zinc	81	1.500	1.00	121.500
Organics				
DRPH	3,190	0.033	1.00	106.756
Ethylbenzene	0.32	0.585	1.00	0.187
Xylenes (total)	1	0.970	1.00	0.970
Tetrachloroethene	1.1	1.336	1.00	1.469
Naphthalene	0.1	0.443	1.00	0.044
Trimethylbenzene	1.4	0.253	1.00	0.354

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS
CF = CS*Bv*%V

SPECIES Lapland longspur COC	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
Inorganics				
Aluminum	0	0.004	0.25	0.000
Cadmium	19	0.550	0.25	2.613
Iron	57,000	0.004	0.25	57.000
Lead	30	0.045	0.25	0.338
Manganese	610	0.250	0.25	38.125
Zinc	81	1.500	0.25	30.375
Organics				
DRPH	3,190	0.033	0.25	26.689
Ethylbenzene	0.32	0.585	0.25	0.047
Xylenes (total)	1	0.970	0.25	0.243
Tetrachloroethene	1.1	1.336	0.25	0.367
Naphthalene	0.1	0.443	0.25	0.011
Trimethylbenzene	1.4	0.253	0.25	0.089

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS
CF = CS*Bv*%V

SPECIES glaucous gull COC	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
Inorganics				
Aluminum	0	0.004	0.10	0.000
Cadmium	19	0.550	0.10	1.045
Iron	57,000	0.004	0.10	22.800
Lead	30	0.045	0.10	0.135
Manganese	610	0.250	0.10	15.250
Zinc	81	1.500	0.10	12.150
Organics				
DRPH	3,190	0.033	0.10	10.676
Ethylbenzene	0.32	0.585	0.10	0.019
Xylenes (total)	1	0.970	0.10	0.097
Tetrachloroethene	1.1	1.336	0.10	0.147
Naphthalene	0.1	0.443	0.10	0.004
Trimethylbenzene	1.4	0.253	0.10	0.035

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS
CF = CS*Bv*%V

SPECIES brant / COC	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
Inorganics				
Aluminum	0	0.004	0.90	0.000
Cadmium	19	0.550	0.90	9.405
Iron	57,000	0.004	0.90	205.200
Lead	30	0.045	0.90	1.215
Manganese	610	0.250	0.90	137.250
Zinc	81	1.500	0.90	109.350
Organics				
DRPH	3,190	0.033	0.90	96.080
Ethylbenzene	0.32	0.585	0.90	0.169
Xylenes (total)	1	0.970	0.90	0.873
Tetrachloroethene	1.1	1.336	0.90	1.322
Naphthalene	0.1	0.443	0.90	0.040
Trimethylbenzene	1.4	0.253	0.90	0.319

CONTAMINANT CONCENTRATION IN FOOD CALCULATIONS

PLANT UPTAKE AND DIETARY PROPORTION OF VEGETATION CALCULATIONS
CF = CS*Bv*%V

SPECIES pectoral sandpiper COC	COC Concentration Soil/Sediment (CS) mg/kg	Bioconcentration Factor (Bv) unitless	Proportion of vegetation in diet (%V) %	COC Concentration in food (CF) mg/kg
Inorganics				
Aluminum	0	0.004	0.10	0.000
Cadmium	19	0.550	0.10	1.045
Iron	57,000	0.004	0.10	22.800
Lead	30	0.045	0.10	0.135
Manganese	610	0.250	0.10	15.250
Zinc	81	1.500	0.10	12.150
Organics				
DRPH	3,190	0.033	0.10	10.676
Ethylbenzene	0.32	0.585	0.10	0.019
Xylenes (total)	1	0.970	0.10	0.097
Tetrachloroethene	1.1	1.336	0.10	0.147
Naphthalene	0.1	0.443	0.10	0.004
Trimethylbenzene	1.4	0.253	0.10	0.035

APPENDIX D

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = ({[(CF*FI) + (CS*SI*ROA) + (CW*WI)] *.001}* IS) / BW

SPECIES arctic fox	COC Conc. Food Items (CF)	Food Intake Rate (FI)	(CF*FI)	Soil/Sed. Intake % (SI%)	Soil/Sed. Ingestion Rate (SI)	COC Conc. Soil /Sed. (CS)	Relative Oral Availability (ROA)	(SI*CS*ROA)	COC Conc. Water (CW)	Water Intake Rate (WI)	(CW*WI)	(A+B+C)	Conver. Units 0.001	Percent Ingested at Site (IS)	Body Weight (BW)	ESTIMATED EXPOSURE (D*IS/BW=EE)
COC	(mg/kg)	(g/day)	(A)	% of FI	(g/day)	(mg/kg)	(unitless)	(B)	(ug/L)	(L/day)	(C)	(D)	(D)*.001	(unitless)	(kg)	(mg/kg-bw/day)
Inorganics																
Aluminum		256	0	0.028	7.168	0	1	0	2000	0.42	840	840	0.84	0.01	4.95	0.002
Cadmium		256	0	0.028	7.168	19	1	136.192	0	0.42	0	136.192	0.136192	0.01	4.95	0.000
Iron		256	0	0.028	7.168	57000	1	408576	28000	0.42	11760	420336	420.336	0.01	4.95	0.849
Lead		256	0	0.028	7.168	30	1	215.04	0	0.42	0	215.04	0.21504	0.01	4.95	0.000
Manganese		256	0	0.028	7.168	610	1	4372.48	690	0.42	289.8	4662.28	4.66228	0.01	4.95	0.009
Vanadium		256	0	0.028	7.168	0	1	0	31	0.42	13.02	13.02	0.01302	0.01	4.95	0.000
Zinc		256	0	0.028	7.168	81	1	580.608	670	0.42	281.4	862.008	0.862008	0.01	4.95	0.002
Organics																
DRPH		256	0	0.028	7.168	3190	1	22865.92		0.42	0	22865.92	22.86592	0.01	4.95	0.046
Ethylbenzene		256	0	0.028	7.168	0.32	1	2.29376		0.42	0	2.29376	0.00229376	0.01	4.95	0.000
Xylenes (total)		256	0	0.028	7.168	1	1	7.168		0.42	0	7.168	0.007168	0.01	4.95	0.000
Tetrachloroethene		256	0	0.028	7.168	1.1	1	7.8848		0.42	0	7.8848	0.0078848	0.01	4.95	0.000
Naphthalene		256	0	0.028	7.168	0.1	1	0.7168		0.42	0	0.7168	0.0007168	0.01	4.95	0.000
Trimethylbenzene		256	0	0.028	7.168	1.4	1	10.0352		0.42	0	10.0352	0.0100352	0.01	4.95	0.000

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = ({[(CF*FI) + (CS*SI*ROA) + (CW*WI)] *.001}* IS) / BW

SPECIES brown lemming / COC	COC Conc. Food Items (CF) (mg/kg)	Food Intake Rate (FI) (g/day)	(CF*FI) (A)	Soil/Sed. Intake % (SI%) % of FI	COC Conc. Soil /Sed. (CS) (mg/kg)	Soil/Sed. Ingestion Rate (SI) (g/day)	Relative Oral Availability (ROA) (unitless)	(CS*SI*ROA) (B)	COC Conc. Water (CW) (ug/L)	Water Intake Rate (WI) (L/day)	(CW*WI) (C)	(A+B+C) (D)	Conver. Units 0.001 (D)*.001	Percent Ingested at Site (IS) (unitless)	Body Weight (BW) (kg)	ESTIMATED EXPOSURE (D*IS/BW=EE) (mg/kg-bw/day)
Inorganics																
Aluminum	0	45	0	0.027	0	1.215	1	0	2000	0.007	14	14	0.014	0.5	0.055	0.127
Cadmium	10.45	45	470.25	0.027	19	1.215	1	23.085	0	0.007	0	493.335	0.493335	0.5	0.055	4.485
Iron	228	45	10260	0.027	57000	1.215	1	69255	28000	0.007	196	79711	79.711	0.5	0.055	724.645
Lead	1.35	45	60.75	0.027	30	1.215	1	36.45	0	0.007	0	97.2	0.0972	0.5	0.055	0.884
Manganese	152.5	45	6862.5	0.027	610	1.215	1	741.15	690	0.007	4.83	7608.48	7.60848	0.5	0.055	69.168
Vanadium	0	45	0	0.027	0	1.215	1	0	31	0.007	0.217	0.217	0.000217	0.5	0.055	0.002
Zinc	121.5	45	5467.5	0.027	81	1.215	1	98.415	670	0.007	4.69	5570.605	5.570605	0.5	0.055	50.642
Organics						0										
DRPH	106.7556043	45	4804.0022	0.027	3190	1.215	1	3875.85		0.007	0	8679.8522	8.67985219	0.5	0.055	78.908
Ethylbenzene	1.469104487	45	66.109702	0.027	0.32	1.215	1	0.3888		0.007	0	66.498502	0.0664985	0.5	0.055	0.605
Xylenes (total)	0.044250685	45	1.9912808	0.027	1	1.215	1	1.215		0.007	0	3.2062808	0.00320628	0.5	0.055	0.029
Tetrachloroethene	0.354232199	45	15.940449	0.027	1.1	1.215	1	1.3365		0.007	0	17.276949	0.01727695	0.5	0.055	0.157
Naphthalene	0	45	0	0.027	0.1	1.215	1	0.1215		0.007	0	0.1215	0.0001215	0.5	0.055	0.001
Trimethylbenzene	0	45	0	0.027	1.4	1.215	1	1.701		0.007	0	1.701	0.001701	0.5	0.055	0.015

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = ({[(CF*FI) + (CS*SI*ROA) + (CW*WI)] *.001}* IS) / BW

SPECIES caribou COC	COC Conc. Food Items (CF) (mg/kg)	Food Intake Rate (FI) (g/day)	(CF*FI) (A)	Soil/Sed. Intake % (SI%) % of FI	COC Conc. Soil /Sed. (CS) (mg/kg)	Soil/Sed. Ingestion Rate (SI) (g/day)	Relative Oral Availability (ROA) (unitless)	(CS*SI*ROA) (B)	COC Conc. Water (CW) (ug/L)	Water Intake Rate (WI) (L/day)	(CW*WI) (C)	(A+B+C) (D)	Conver. Units 0.001 (D)*.001	Percent Ingested at Site (IS) (unitless)	Body Weight (BW) (kg)	ESTIMATED EXPOSURE (D*IS/BW=EE) (mg/kg-bw/day)
Inorganics																
Aluminum	0	2400	0	0.02	0	48	1	0	2000	6.0	12000	12000	12	0.01	95.5	0.001
Cadmium	10.45	2400	25080	0.02	19	48	1	912	0	6.0	0	25992	25.992	0.01	95.5	0.003
Iron	228	2400	547200	0.02	57000	48	1	2736000	28000	6.0	168000	3451200	3451.2	0.01	95.5	0.361
Lead	1.35	2400	3240	0.02	30	48	1	1440	0	6.0	0	4680	4.68	0.01	95.5	0.000
Manganese	152.5	2400	366000	0.02	610	48	1	29280	690	6.0	4140	399420	399.42	0.01	95.5	0.042
Vanadium		2400	0	0.02	0	48	1	0	31	6.0	186	186	0.186	0.01	95.5	0.000
Zinc	121.5	2400	291600	0.02	81	48	1	3888	670	6.0	4020	299508	299.508	0.01	95.5	0.031
Organics																
DRPH	106.7556043	2400	256213.45	0.02	3190	48	1	153120		6.0	0	409333.45	409.33345	0.01	95.5	0.043
Ethylbenzene	1.469104487	2400	3525.8508	0.02	0.32	48	1	15.36		6.0	0	3541.2108	3.54121077	0.01	95.5	0.000
Xylenes (total)	0.044250685	2400	106.20164	0.02	1	48	1	48		6.0	0	154.20164	0.15420164	0.01	95.5	0.000
Tetrachloroethene	0.354232199	2400	850.15728	0.02	1.1	48	1	52.8		6.0	0	902.95728	0.90295728	0.01	95.5	0.000
Naphthalene	0	2400	0	0.02	0.1	48	1	4.8		6.0	0	4.8	0.0048	0.01	95.5	0.000
Trimethylbenzene	0	2400	0	0.02	1.4	48	1	67.2		6.0	0	67.2	0.0672	0.01	95.5	0.000

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = ({[(CF*FI) + (CS*SI*ROA) + (CW*WI)] *.001}* IS) / BW

SPECIES Lapland longspur COC	COC Conc. Food Items (CF) (mg/kg)	Food Intake Rate (FI) (g/day)	(CF*FI) (A)	Soil/Sed. Intake % (SI%) % of FI	COC Conc. Soil /Sed. (CS) (mg/kg)	Soil/Sed. Ingestion Rate (SI) (g/day)	Relative Oral Availability (ROA) (unitless)	(CS*SI*ROA) (B)	COC Conc. Water (CW) (ug/L)	Water Intake Rate (WI) (L/day)	(CW*WI) (C)	(A+B+C) (D)	Conver. Units 0.001 (D)*.001	Percent Ingested at Site (IS) (unitless)	Body Weight (BW) (kg)	ESTIMATED EXPOSURE (D*IS/BW=EE) (mg/kg-bw/day)
Inorganics																
Aluminum	0	6.6	0	0.02	0	0.132	1	0	2000	0.005	10	10	0.01	0.5	0.027	0.185
Cadmium	2.6125	6.6	17.2425	0.02	19	0.132	1	2.508	0	0.005	0	19.7505	0.0197505	0.5	0.027	0.366
Iron	57	6.6	376.2	0.02	57000	0.132	1	7524	28000	0.005	140	8040.2	8.0402	0.5	0.027	148.893
Lead	0.3375	6.6	2.2275	0.02	30	0.132	1	3.96	0	0.005	0	6.1875	0.0061875	0.5	0.027	0.115
Manganese	38.125	6.6	251.625	0.02	610	0.132	1	80.52	690	0.005	3.45	335.595	0.335595	0.5	0.027	6.215
Vanadium		6.6	0	0.02	0	0.132	1	0	31	0.005	0.155	0.155	0.000155	0.5	0.027	0.003
Zinc	30.375	6.6	200.475	0.02	81	0.132	1	10.692	670	0.005	3.35	214.517	0.214517	0.5	0.027	3.973
Organics																
DRPH	26.68890106	6.6	176.14675	0.02	3190	0.132	1	421.08		0.005	0	597.22675	0.59722675	0.5	0.027	11.060
Ethylbenzene	0.367276122	6.6	2.4240224	0.02	0.32	0.132	1	0.04224		0.005	0	2.4662624	0.00246626	0.5	0.027	0.046
Xylenes (total)	0.011062671	6.6	0.0730136	0.02	1	0.132	1	0.132		0.005	0	0.2050136	0.00020501	0.5	0.027	0.004
Tetrachloroethene	0.08855805	6.6	0.5844831	0.02	1.1	0.132	1	0.1452		0.005	0	0.7296831	0.00072968	0.5	0.027	0.014
Naphthalene	0	6.6	0	0.02	0.1	0.132	1	0.0132		0.005	0	0.0132	0.0000132	0.5	0.027	0.000
Trimethylbenzene	0	6.6	0	0.02	1.4	0.132	1	0.1848		0.005	0	0.1848	0.0001848	0.5	0.027	0.003

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = ({[(CF*FI) + (CS*SI*ROA) + (CW*WI)] *.001}* IS) / BW

SPECIES glaucous gull COC	COC Conc. Food Items (CF) (mg/kg)	Food Intake Rate (FI) (g/day)	(CF*FI) (A)	Soil/Sed. Intake % (SI%) % of FI	COC Conc. Soil /Sed. (CS) (mg/kg)	Soil/Sed. Ingestion Rate (SI) (g/day)	Relative Oral Availability (ROA) (unitless)	(CS*SI*ROA) (B)	COC Conc. Water (CW) (ug/L)	Water Intake Rate (WI) (L/day)	(CW*WI) (C)	(A+B+C) (D)	Conver. Units 0.001 (D)*.001	Percent Ingested at Site (IS) (unitless)	Body Weight (BW) (kg)	ESTIMATED EXPOSURE (D*IS/BW=EE) (mg/kg-bw/day)
Inorganics																
Aluminum	0	73.9	0	0.076	0	5.6164	1	0	2000	0.08	160	160	0.16	0.04	1.45	0.004
Cadmium	1.045	73.9	77.2255	0.076	19	5.6164	1	106.7116	0	0.08	0	183.9371	0.1839371	0.04	1.45	0.005
Iron	22.8	73.9	1684.92	0.076	57000	5.6164	1	320134.8	28000	0.08	2240	324059.72	324.05972	0.04	1.45	8.940
Lead	0.135	73.9	9.9765	0.076	30	5.6164	1	168.492	0	0.08	0	178.4685	0.1784685	0.04	1.45	0.005
Manganese	15.25	73.9	1126.975	0.076	610	5.6164	1	3426.004	690	0.08	55.2	4608.179	4.608179	0.04	1.45	0.127
Vanadium		73.9	0	0.076	0	5.6164	1	0	31	0.08	2.48	2.48	0.00248	0.04	1.45	0.000
Zinc	12.15	73.9	897.885	0.076	81	5.6164	1	454.9284	670	0.08	53.6	1406.4134	1.4064134	0.04	1.45	0.039
Organics																
DRPH	10.67556043	73.9	788.92392	0.076	3190	5.6164	1	17916.316		0.08	0	18705.24	18.7052399	0.04	1.45	0.516
Ethylbenzene	0.146910449	73.9	10.856682	0.076	0.32	5.6164	1	1.797248		0.08	0	12.65393	0.01265393	0.04	1.45	0.000
Xylenes (total)	0.004425069	73.9	0.3270126	0.076	1	5.6164	1	5.6164		0.08	0	5.9434126	0.00594341	0.04	1.45	0.000
Tetrachloroethene	0.03542322	73.9	2.617776	0.076	1.1	5.6164	1	6.17804		0.08	0	8.795816	0.00879582	0.04	1.45	0.000
Naphthalene	0	73.9	0	0.076	0.1	5.6164	1	0.56164		0.08	0	0.56164	0.00056164	0.04	1.45	0.000
Trimethylbenzene	0	73.9	0	0.076	1.4	5.6164	1	7.86296		0.08	0	7.86296	0.00786296	0.04	1.45	0.000

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = ({[(CF*FI) + (CS*SI*ROA) + (CW*WI)] *.001}* IS) / BW

SPECIES brant / COC	COC Conc. Food Items (CF) (mg/kg)	Food Intake Rate (FI) (g/day)	(CF*FI) (A)	Soil/Sed. Intake % (SI%) % of FI	COC Conc. Soil /Sed. (CS) (mg/kg)	Soil/Sed. Ingestion Rate (SI) (g/day)	Relative Oral Availability (ROA) (unitless)	(CS*SI*ROA) (B)	COC Conc. Water (CW) (ug/L)	Water Intake Rate (WI) (L/day)	(CW*WI) (C)	(A+B+C) (D)	Conver. Units 0.001 (D)*.001	Percent Ingested at Site (IS) (unitless)	Body Weight (BW) (kg)	ESTIMATED EXPOSURE (D*IS/BW=EE) (mg/kg-bw/day)
Inorganics																
Aluminum	0	69.2	0	0.082	0	5.6744	1	0	2000	0.07	140	140	0.14	0.25	1.31	0.027
Cadmium	9.405	69.2	650.826	0.082	19	5.6744	1	107.8136	0	0.07	0	758.6396	0.7586396	0.25	1.31	0.145
Iron	205.2	69.2	14199.84	0.082	57000	5.6744	1	323440.8	28000	0.07	1960	339600.64	339.60064	0.25	1.31	64.809
Lead	1.215	69.2	84.078	0.082	30	5.6744	1	170.232	0	0.07	0	254.31	0.25431	0.25	1.31	0.049
Manganese	137.25	69.2	9497.7	0.082	610	5.6744	1	3461.384	690	0.07	48.3	13007.384	13.007384	0.25	1.31	2.482
Vanadium		69.2	0	0.082	0	5.6744	1	0	31	0.07	2.17	2.17	0.00217	0.25	1.31	0.000
Zinc	109.35	69.2	7567.02	0.082	81	5.6744	1	459.6264	670	0.07	46.9	8073.5464	8.0735464	0.25	1.31	1.541
Organics																
DRPH	96.08004383	69.2	6648.739	0.082	3190	5.6744	1	18101.336		0.07	0	24750.075	24.750075	0.25	1.31	4.723
Ethylbenzene	1.322194038	69.2	91.495827	0.082	0.32	5.6744	1	1.815808		0.07	0	93.311635	0.09331164	0.25	1.31	0.018
Xylenes (total)	0.039825617	69.2	2.7559327	0.082	1	5.6744	1	5.6744		0.07	0	8.4303327	0.00843033	0.25	1.31	0.002
Tetrachloroethene	0.318808979	69.2	22.061581	0.082	1.1	5.6744	1	6.24184		0.07	0	28.303421	0.02830342	0.25	1.31	0.005
Naphthalene	0	69.2	0	0.082	0.1	5.6744	1	0.56744		0.07	0	0.56744	0.00056744	0.25	1.31	0.000
Trimethylbenzene	0	69.2	0	0.082	1.4	5.6744	1	7.94416		0.07	0	7.94416	0.00794416	0.25	1.31	0.002

ESTIMATED EXPOSURE EQUATION FOR BIRDS AND MAMMALS

Estimated Exposure = ({[(CF*FI) + (CS*SI*ROA) + (CW*WI)] *.001}* IS) / BW

SPECIES pectoral sandpiper / COC	COC Conc. Food Items (CF) (mg/kg)	Food Intake Rate (FI) (g/day)	(CF*FI) (A)	Soil/Sed. Intake % (SI%) % of FI	COC Conc. Soil /Sed. (CS) (mg/kg)	Soil/Sed. Ingestion Rate (SI) (g/day)	Relative Oral Availability (ROA) (unitless)	(CS*SI*ROA) (B)	COC Conc. Water (CW) (ug/L)	Water Intake Rate (WI) (L/day)	(CW*WI) (C)	(A+B+C) (D)	Conver. Units 0.001 (D)*.001	Percent Ingested at Site (IS) (unitless)	Body Weight (BW) (kg)	ESTIMATED EXPOSURE (D*IS/BW=EE) (mg/kg-bw/day)
Inorganics																
Aluminum	0	11.1	0	0.181	0	2.0091	1	0	2000	0.01	20	20	0.02	1.0	0.08	0.250
Cadmium	1.045	11.1	11.5995	0.181	19	2.0091	1	38.1729	0	0.01	0	49.7724	0.0497724	1.0	0.08	0.622
Iron	22.8	11.1	253.08	0.181	57000	2.0091	1	114518.7	28000	0.01	280	115051.78	115.05178	1.0	0.08	1438.147
Lead	0.135	11.1	1.4985	0.181	30	2.0091	1	60.273	0	0.01	0	61.7715	0.0617715	1.0	0.08	0.772
Manganese	15.25	11.1	169.275	0.181	610	2.0091	1	1225.551	690	0.01	6.9	1401.726	1.401726	1.0	0.08	17.522
Vanadium		11.1	0	0.181	0	2.0091	1	0	31	0.01	0.31	0.31	0.00031	1.0	0.08	0.004
Zinc	12.15	11.1	134.865	0.181	81	2.0091	1	162.7371	670	0.01	6.7	304.3021	0.3043021	1.0	0.08	3.804
Organics																
DRPH	10.67556043	11.1	118.49872	0.181	3190	2.0091	1	6409.029		0.01	0	6527.5277	6.52752772	1.0	0.08	81.594
Ethylbenzene	0.146910449	11.1	1.630706	0.181	0.32	2.0091	1	0.642912		0.01	0	2.273618	0.00227362	1.0	0.08	0.028
Xylenes (total)	0.004425069	11.1	0.0491183	0.181	1	2.0091	1	2.0091		0.01	0	2.0582183	0.00205822	1.0	0.08	0.026
Tetrachloroethene	0.03542322	11.1	0.3931977	0.181	1.1	2.0091	1	2.21001		0.01	0	2.6032077	0.00260321	1.0	0.08	0.033
Naphthalene	0	11.1	0	0.181	0.1	2.0091	1	0.20091		0.01	0	0.20091	0.00020091	1.0	0.08	0.003
Trimethylbenzene	0	11.1	0	0.181	1.4	2.0091	1	2.81274		0.01	0	2.81274	0.00281274	1.0	0.08	0.035

APPENDIX E

BIOCONCENTRATION FACTOR CALCULATIONS FOR ORGANIC CHEMICALS

BIOCONCENTRATION FACTOR CALCULATIONS for ORGANIC CHEMICALS

CALCULATION OF Bv FOR ORGANIC CHEMICALS IN SOIL

COC Organics	log Kow	1.588 - 0.578 log Kow	log Bv	Bv
DRPH	5.30	-1.475	-1.475	0.033
Ethylbenzene	3.15	-0.233	-0.233	0.585
Xylenes (total)*	2.77	-0.013	-0.013	0.970
Tetrachloroethene	2.53	0.126	0.126	1.336
Napthalene	3.36	-0.354	-0.354	0.443
Trimethylbenzene	3.78	-0.597	-0.597	0.253

* log Kow for ortho-xylene used

APPENDIX F

SCALING FACTOR CALCULATIONS

SCALING FACTOR CALCULATIONS

Scaling factor (SF) = (representative species average body weight/ test species average body weight)$^{1/3}$ based on the mass to surface area ratios of the test species and the representative species (Mantel and Scheiderman 1975)

Representative Species	Average Body Weight[1] (kg)	Test Species	Average Body Weight[2] (kg)	Scaling Factor (SF)
brown lemming	0.055	mouse	0.025	1.30
	0.055	rat	0.25	0.60
arctic Fox	4.95	rat	0.25	2.70
	4.95	mouse	0.025	5.82
caribou	95.5	sheep	60	1.17
	95.5	cattle	500	0.58
	95.5	rat	0.25	7.24
	95.5	mouse	0.025	15.59
Lapland longspur	0.027	chicken	0.8	0.32
	0.027	mallard	1.08	0.29
	0.027	Japanese quail	0.10	0.65
	0.027	ringed dove	0.155	0.56
brant	1.305	chicken	0.8	1.18
	1.305	mallard	1.08	1.07
	1.305	Japanese quail	0.10	2.35
	1.305	ringed dove	0.155	2.03
glaucous gull	1.445	chicken	0.8	1.22
	1.445	mallard	1.08	1.10
	1.445	Japanese quail	0.10	2.43
	1.445	ringed dove	0.155	2.10
pectoral sandpiper	0.079	chicken	0.8	0.46
	0.079	mallard	1.08	0.42
	0.079	Japanese quail	0.10	0.92
	0.079	ringed dove	0.155	0.80

[1]average body weight for the representative species from the Life History Information Tables
[2] From Sax and Lewis (1989)

APPENDIX G

DATA SUMMARY TABLES

TABLE G-1. SUMMARY OF SAMPLING AND ANALYSES CONDUCTED FOR WA

ANALYSES	HVOC*	BTEX*	VOC 8260	SVOC	Metals[b]	TPH-Diesel[b] Range 3510/3550	TPH - Gasoline[b] Range	TPI Resic Rang
ANALYTICAL METHOD	SW8010M	SW8020	SW8260	SW8270	SW3050 (Soil) 3005 (Water)/6010	Diesel 8100M	Gas 5030/8015M	Diesel 8
WAINWRIGHT (LIZ-3)								
Background (BKGD)	4 Soil 2 Water	4 Soil 2 Water	4 Soil 2 Water	4 Soil 2 Water	4 Soil 2 Water (Total) 2 Water (Dissolved)	4 Soil 2 Water	4 Soil 2 Water	4 S 2 Wa
Drum Storage Area (ST02)	5 Soil	5 Soil	2 Soil	2 Soil	2 Soil	5 Soil	5 Soil	5 S
Diesel Fuel Spills (SS04)	NA	9 Soil 3 Water	2 Soil 1 Water	1 Water	NA	12 Soil 3 Water	10 Soil 3 Water	11 S 3 Wa
Landfill (LF05)	6 Soil 2 Water	6 Soil 2 Water	2 Soil 2 Water	2 Soil 2 Water	2 Soil 2 Water (Total) 2 Water (Dissolved)	6 Soil 2 Water	6 Soil 2 Water	6 S 2 Wa
Garage (SS07)	6 Soil	6 Soil 3 Water	2 Soil 2 Water	2 Water	2 Soil 2 Water (Total) 2 Water (Dissolved)	9 Soil 5 Water	7 Soil 3 Water	8 S 5 Wa
Airstrip Diesel Spill (SS08)	NA	4 Soil 3 Water	1 Soil 1 Water	1 Water	NA	4 Soil 3 Water	4 Soil 3 Water	4 S 3 Wa
Vehicle Storage Area (SS09)[c]	11 Soil 2 Water	11 Soil 2 Water	4 Soil 2 Water	2 Soil 2 Water	4 Soil 2 Water (Total) 2 Water (Dissolved)	11 Soil 2 Water	11 Soil 2 Water	11 S 2 Wa
Total Field Analyses	32 Soil 6 Water	45 Soil 15 Water	17 Soil 10 Water	10 Soil 10 Water	14 Soil 8 Water (Total) 8 Water (Dissolved)	51 Soil 17 Water	47 Soil 15 Water	49 S 17 W
QA/QC SAMPLES								
Trip Blanks	3 Water	3 Water	3 Water	NA	NA	NA	3 Water	NA
Equipment Blanks	3 Water	3 Water	2 Water	2 Water	3 Water (Total)	3 Water	3 Water	3 Wa
Ambient Condition Blanks	1 Water	1 Water	NA	NA	NA	NA	1 Water	[illegible]
Field Replicates	3 Soil	4 Soil	2 Soil	1 Soil	1 Soil	5 Soil	4 Soil	5 S
Field Duplicates	NA	2 Water	1 Water	1 Water	NA	3 Water	2 Water	3 W
Total Site Analyses	35 Soil 10 Water	49 Soil 24 Water	19 Soil 16 Water	11 Soil 13 Water	15 Soil 11 Water (Total) 8 Water (Dissolved)	56 Soil 23 Water	51 Soil 24 Water	54 S 23 W

NA Not analyzed.
* These analyses were completed on a quick turnaround basis.
a The number of soil sample includes sediment samples collected from surface water features.
b Some of these analysis were completed on a 24-hour turnaround at a temporary fixed laboratory at Barrow, Alaska.
c Stockpiled soils located in the vicinity of the Vehicle Storage Area were sampled in conjunction with this site.
d Investigation derived wastes from Wainwright were combined with the investigation derived wastes from Point Lay, Point Barrow, an

AK-RISK\WAINWR\APP-G\G-1.TBL

CONDUCTED FOR WAINWRIGHT REMEDIAL INVESTIGATIONS[a]

TPH - Gasoline[b] Range	TPH Residual Range*	PCB*	Pesticides*	TDS	TSS	TOC	TCLP[d]	TOTAL SAMPLES
Gas 5030/8015M	Diesel 8100M	SW8080/8080M	SW8080/8080M	E160.1	E160.2	SW9060	SW1311	
4 Soil 2 Water	4 Soil 2 Water	4 Soil 2 Water	4 Soil 2 Water	2 Water	2 Water	4 Soil 1 Water	NA	4 Soil 2 Water
5 Soil	5 Soil	5 Soil	NA	NA	NA	NA	NA	5 Soil
10 Soil 3 Water	11 Soil 3 Water	5 Soil	2 Soil	1 Water	1 Water	1 Water	NA	11 Soil 3 Water
6 Soil 2 Water	6 Soil 2 Water	6 Soil 2 Water	1 Soil	2 Water	2 Water	2 Soil 2 Water	NA	6 Soil 2 Water
7 Soil 3 Water	8 Soil 5 Water	6 Soil	3 Soil 2 Water	NA	NA	NA	NA	8 Soil 5 Water
4 Soil 3 Water	4 Soil 3 Water	NA	1 Water	1 Water	1 Water	1 Water	NA	4 Soil 3 Water
11 Soil 2 Water	11 Soil 2 Water	7 Soil 2 Water	1 Water	2 Water	2 Water	2 Soil 1 Water	NA	11 Soil 2 Water
47 Soil 15 Water	49 Soil 17 Water	33 Soil 6 Water	10 Soil 6 Water	8 Water	8 Water	8 Soil 6 Water	NA	49 Soil 17 Water
3 Water	NA	NA	NA	NA	NA	NA	NA	3 Water
3 Water	3 Water	3 Water	3 Water	NA	NA	3 Water	NA	3 Water
1 Water	NA	NA	NA	NA	NA	NA	NA	1 Water
4 Soil	5 Soil	4 Soil	NA	NA	NA	NA	NA	5 Soil
2 Water	3 Water	NA	NA	1 Water	1 Water	1 Water	NA	3 Water
51 Soil 24 Water	54 Soil 23 Water	37 Soil 9 Water	10 Soil 9 Water	9 Water	9 Water	8 Soil 11 Water	1 Water	54 Soil 27 Water

ow, Alaska.
site.
Point Lay, Point Barrow, and Point Lonely. These were collectively sampled during the Point Barrow investigation.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY

Installation: Wainwright
Site: Background (BKGD)
Matrix: Soil/Sediment
Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples				Field Blanks				Lab Blanks	
					S01-0.5	SD01-0.5	2S02-1	2SD02-1	AB01	EB01	TB01	2EB03		
Laboratory Sample ID Numbers					1236 4479-6	1238 4479-5	1876 4694-4	1874 4694-1	1424	1280/1282 4479-7	1260 4479-8	1894/1896 4695-3	#6-9993 #5-9193 #1&2-9793 #1&2-9293 4695 4479	#6-91093 #6-83193 #3&4-9293 #1&2-91093 4694 4479
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	5-30	50-300	500[a]	<50[b]-<300[b]	<50[b]	<60[b]	<75[b]	<300J[b]	NA	<1,000[b]	NA	<1,000J[b]	<1,000	<50J-<50
GRPH	0.1-0.5	1-5	100	<2J[b]-<5J[b]	<2J[b]	<2J[b]	<1J[b]	<5J[b]	<50J[b]	<100J[b]	<100J[b]	<50J[b]	<50J	<1-<2J
RRPH (Approx.)	10-60	100-600	2,000[a]	<100-<600	<100	<120	<200	<600	NA	<2,000	NA	<2,000	<2,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.10-<0.5	<0.10	<0.10	<0.15	<0.5						
Benzene	0.002-0.01	0.02-0.1	0.5	<0.02-<0.1	<0.02	<0.02	<0.03	<0.1	<1	<1	<1	<1	<1-<5	<0.02
Toluene	0.002-0.01	0.02-0.1		<0.02-<0.1	<0.02	<0.02	<0.03	<0.1	<1	<1	<1	<1	<1	<0.02
Ethyl-benzene	0.002-0.01	0.02-0.1		<0.02-<0.1	<0.02	<0.02	<0.03	<1	<1	<1	<1	<1	<1	<0.02
Xylenes (Total)	0.004-0.02	0.04-0.2		<0.04-<0.2	<0.04	<0.04	<0.06	<0.2	<2	<2	<2	<2	<2	<0.04
HVOC 8010	0.002-0.05	0.02-0.5		<0.02-<0.5	<0.02	<0.02	<0.1J	<0.5J	<1	<10	<10	<5	<1-<10J	<0.02
VOC 8260	0.020	0.020-0.400		<0.020-<0.400	<0.020	<0.025	<0.060	<0.400J	NA	<1	<1	<1-4.1	<1	<0.020
SVOC 8270														
di-n-Butyl phthalate	0.200	0.200-32.0		1.69U-83.4J	1.69U	2.03B	9.01B	83.4J	NA	<10	NA	<21	<10	0.741-1.41

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
B Analyte was found in the associated blank.
J Result is an estimate.
U Compound is not present above concentration listed.
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Background (BKGD)
Matrix: Soil/Sediment
Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples				Field Blanks				Lab Blanks	
					S01-0.5	SD01-0.5	2S02-1	2SD02-1	AB01	EB01	TB01	2EB03		
Laboratory Sample ID Numbers					1236 4479-6	1238 4479-5	1876 4694-4	1874 4694-1	1424	1280/1[illegible] 44[illegible]	1260 4479-8	1894/1896 4695-3	#6-9993 #5-9193 4695 4479	#6-91093 #6-83193 4694 4479
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	[illegible]	µg/L	µg/L	µg/L	µg/L
Pesticides	0.001-0.28	0.01-2.8		<0.01J-2.8J	<0.01J-<0.5J	<0.01J-<0.5J	<0.02J-<0.80J	<0.08J-<2.8J	NA	<0.2J-<10J	NA	<0.2J-<25J	NA	<0.01
PCBs	0.01-0.28	0.1-2.8	10	<0.1-<2.8J	<0.1	<0.1	<0.8J	<2.8J	NA	<2J	NA	<2J	<2	<0.1
TOC				10,500-44,100	10,500	43,300	44,100	19,400	NA	<5,000	NA	<5,000	<5,000	NA

☐	CT&E Data.
▩	F&B Data.
NA	Not analyzed.
J	Result is an estimate.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright　Matrix: Soil/Sediment　METALS ANALYSES
Site: Background (BKGD)　Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Bkgd. Range from 7 DEW Line Installations	Wainwright Bkgd. Range	Environmental Samples						Field Blanks		Lab Blanks
					S01-0.5	SD01-0.5	2S02-1	2SD02-1			EB01	2EB03	
Laboratory Sample ID Numbers					4479-6	4479-5	4694-4	4694-1			4479-7	4695-3	4479 4695 4694
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg			µg/L	µg/L	µg/L
Aluminum	0.35	2	1,500-25,000	1,500-5,900	1,500	1,600	5,900	1,500			<100	<100	<100
Antimony	N/A	7.8-54	<7.8-<230	<7.8-<54	<51J	<54	<7.8	<14			<100	<100	<100
Arsenic	0.11	7.8-51	<4.9-8.5	<5.4-<51	<51	<5.4	<7.8	<14			<100	<100	<100
Barium	0.024	1	27-390	62-120	110	62	120	81			<50	<50	<50
Beryllium	N/A	2.6-71	<2.6-6.4	<2.6-<71	<2.6	<2.7	<3.9	<71			<50	<50	<50
Cadmium	0.33	3.9-71	<3.0-<36	<3.9-<27	<26	<27	<3.9	<7.1			<50	<50	<50
Calcium	0.69	4	360-59,000	360-2,400	720	360	690	2,400J			410	<200	<200
Chromium	0.066	1-7.1	<4.3-47	<7.1-10	<26	<27	10	<7.1			<50	<50	<50
Cobalt	N/A	5.1-14	<5.1-12	<5.1-<14	<5.1	<5.4	<7.8	<14			<100	<100	<100
Copper	0.045	1-7.1	<2.7-45	<2.7-7.2	3.8	<2.7	7.2	<7.1			<50	<50	<50
Iron	0.50	2	5,400-35,000	5,400-16,000	13,000	5,600	5,400	16,000			<100	<100	<100
Lead	0.13	5.1-14	<5.1-22	<5.1-<14	<5.1	<5.4	<7.8	<14			<100	<100	<100
Magnesium	0.96	4	360-7,400	360-1,200	360	400	1,200	510			<200	<200	<200
Manganese	0.025	1	25-290	25-44J	44J	25	29	28			<50	<50	<50
Molybdenum	N/A	2.6-7.1	<2.5-<11	<2.6-<7.1	<2.6	<2.7	<3.9	<7.1			<50	<50	<50
Nickel	0.11	1	4.2-46	4.4-8.9	5.6	4.4	7.6	8.9			<50	<50	<50

☐ CT&E Data.
N/A Not available.
J Result is an estimate.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Background (BKGD)

Matrix: Soil/Sediment
Units: mg/kg

METALS ANALYSES

Parameters	Detect. Limits	Quant. Limits	Bkgd. Range from 7 DEW Line Installations	Wainwright Bkgd. Range	Environmental Samples						Field Blanks		Lab Blanks
					S01-0.5	SD01-0.5	2S02-1	2SD02-1			EB01	2EB03	
Laboratory Sample ID Numbers					4479-6	4479-5	4694-4	4694-1			4479-7	4695-3	4479 4695 4694
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg			µg/L	µg/L	µg/L
Potassium	23	100-710	<300-2,200	<710-540	400J	350	540	<710			<5,000	<5,000	<5,000
Selenium	1.2	7.8-5.4	<7.8-<170	<7.8-<54	<51	<54	<7.8	<14			<100	<100	<100
Silver	0.53	3.9-71	<3-<110	<3.9-<71J	<26R	<27	<3.9	<71J			<50R	<50	<50
Sodium	0.55	5	<160-680	41-140	58	41	47	140			410	370	<250
Thallium	0.011	0.25-0.76	<0.2-<1.2	<0.25-<0.76	<0.25	<0.27	<0.38	<0.76			<5	<5	<5
Vanadium	0.036	1	6.3-59	9.5-16	14	9.5	16	9.9			<50	<50	<50
Zinc	0.16	1	9.2-95	9.2-16	13	9.2	14	16			<50	<50	<50

☐ CT&E Data.
J Result is an estimate.
R Result has been rejected.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Background (BKGD)

Matrix: Surface Water
Units: µg/L

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples		Field Blanks				Lab Blanks
					SW01	2SW02	AB01	EB01	TB01	2EB03	
Laboratory Sample ID Numbers					1284/1286 4480-3	1869/1870 1904 4694-8	1424	1280/1282 4479-7	1260 4479-8	1876/1896 4695-3	#6-9993 #5-9193 #1&2-9793 #1&2-9293 4694/4695 4479/4480
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
DRPH	100	1,000		<1,000J[b]	<1,000[b]	<1,000J[b]	NA	<1,000[b]	NA	<1,000J[b]	<1,000J
GRPH	5-10	50-100		<50J[b]-<100J[b]	<100J[b]	<50J[b]	<50J[b]	<100J[b]	<100J[b]	<50J[b]	<50J
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000	NA	<2,000	NA	<2,000	<2,000
BTEX (8020/8020 Mod.)											
Benzene	0.1	1	5	<1	<1	<1	<1	<1	<1	<1	<1-<5
Toluene	0.1	1	1,000	<1	<1	<1	<1	<1	<1	<1	<1
Ethylbenzene	0.1	1	700	<1	<1	<1	<1	<1	<1	<1	<1
Xylenes (Total)	0.2	2	10,000	<2	<2	<2	<2	<2	<2	<2	<2
HVOC 8010	0.5-1	5-10		<5-<10	<10	<5	<1	<10	<10	<5	<1-<10J
VOC 8260	1	1		<1	<1	<1	NA	<1	<1	<1-4.1	<1
SVOC 8270	10	10-13		<10-<13	<10	<13	NA	<10	NA	<21	<10
Pesticides	0.02-2.5	0.2-25		<0.2J-<25J	<0.2J-10J	<0.2J-<25J	NA	<0.2J-<10J	NA	<0.2J-<25J	<0.01J-<0.5J
PCBs	0.2	2	0.5	<2J	<2J	<2J	NA	<2J	NA	<2J	<2J

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Background (BKGD)

Matrix: Surface Water
Units: µg/L

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range	Environmental Samples		Field Blanks				Lab Blanks
					SW01	2SW02	AB01	EB01	TB01	2EB03	
Laboratory Sample ID Numbers					1284/1286 4480-3	1869/1870 1904 4694-8	1424	1280/1282 4479-7	1260 4479-8	1876/1896 4695-3	4694/4695 4479/4480
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
TOC	5,000	5,000		7,480	7,480	NA	NA	<5,000	NA	<5,000	<5,000
TSS	100	200		7,000-35,000	35,000	7,000	NA	NA	NA	NA	<200
TDS	10,000	10,000		91,000-151,000	91,000	151,000	NA	NA	NA	NA	<10,000

☐ CT&E Data.
NA Not analyzed.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright Matrix: Surface Water
Site: Background (BKGD) Units: µg/L METALS ANALYSES: TOTAL (DISSOLVED)

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Wainwright Bkgd. Levels	Environmental Samples				Field Blanks		Lab Blanks
						SW01	2SW02			EB01	2EB03	
Laboratory Sample ID Numbers						4480-3	4694-8			4479-7	4695-3	4694/4695 4479/4480
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L			µg/L	µg/L	µg/L
Aluminum	17.4	100		<100-350 (<100-340)	120-130 (<100)	120 (<100)	130 (<100)			<100	<100	<100
Antimony	N/A	100	6	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)			<100	<100	<100
Arsenic	5.3	100	50	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)			<100	<100	<100
Barium	1.2	50	2,000	<50-93 (<50-91)	<50-52 (<50)	52 (<50)	<50 (<50)			<50	<50	<50
Beryllium	N/A	50	4	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)			<50	<50	<50
Cadmium	1.7	50	5	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)			<50	<50	<50
Calcium	34.5	200		4,500-88,000 (4,100-86,000)	4,500-8,200 (4,100-8,200)	8,200 (8,200)	4,500 (4,100)			410	<200	<200
Chromium	3.29	50	100	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)			<50	<50	<50
Cobalt	N/A	100		<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)			<100	<100	<100
Copper	2.3	50	1,300	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)			<50	<50	<50
Iron	25	100		180-2,800 (<100-1,600)	900-1,200 (190-630)	900 (190)	1,200 (630)			<100	<100	<100
Lead	6.6	100	15	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)			<100	<100	<100

☐ CT&E Data.
N/A Not available.

TABLE G-2. BACKGROUND ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright — Matrix: Surface Water

Site: Background (BKGD) — Units: µg/L — METALS ANALYSES: TOTAL (DISSOLVED)

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Wainwright Bkgd. Levels	Environmental Samples				Field Blanks		Lab Blanks
						SW01	2SW02			EB01	2EB03	
Laboratory Sample ID Numbers						4480-3	4694-8			4479-7	4695-3	4694/4695 4479/4480
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L			µg/L	µg/L	µg/L
Magnesium	47.8	200		<5,000-53,000 (2,600-54,000)	2,900-3,500 (2,600-3,400)	3,500 (3,400)	2,900 (2,600)			<200	<200	<200
Manganese	1.24	50		<50-510 (<50-120)	<50 (<50)	<50 (<50)	<50 (<50)			<50	<50	<50
Molybdenum	N/A	50		<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)			<50	<50	<50
Nickel	5.5	50	100	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)			<50	<50	<50
Potassium	1,154	5,000		<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)			<5,000	<5,000	<5,000
Selenium	62.4	100	50	<100 (<100)	<100 (<100)	<100 (<100)	<100 (<100)			<100	<100	<100
Silver	2.6	50	50	<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)			<50R	<50	<50
Sodium	27.7	250		8,400-410,000 (8,200-450,000)	8,400-9,900 (8,200-10,000)	9,900 (10,000)	8,400 (8,200)			410	370	<250
Thallium	0.57	5	2	<5 (<5)	<5 (<5)	<5 (<5)	<5 (<5)			<5	<5	<5
Vanadium	1.8	50		<50 (<50)	<50 (<50)	<50 (<50)	<50 (<50)			<50	<50	<50
Zinc	8.2	50		<50-160 (<50)	<50 (<50)	<50 (<50)	<50 (<50)			<50	<50	<50

☐ CT&E Data.
N/A Not available.
R Result has been rejected.

TABLE G-3. DRUM STORAGE AREA ANALYTICAL DATA SUMMARY

Installation: Wainwright   Matrix: Soil
Site: Drum Storage Area (ST02)   Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples S01	S02	S03 & S06 (Replicates)		S04	S05	Field Blanks AB01	EB01	TB01	Lab Blanks	
Laboratory Sample ID Numbers					1210	1212 4479-4	1214	1220	1216 4479-3	1218	1424	1280/1282 4479-7	1260 4479-8	#5-9193 #1&2-9293	4479 #6-83193 #3&4-9293
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	5-7	50-70	500[a]	<50[b]-<300J[b]	<70[b]	<60[b]	<70[b]	<60[b]	<60[b]	<50[b]	NA	<1,000[b]	NA	<1,000J	<50
GRPH	0.2	2	100	<2J[b]-<5J[b]	<2J[b]	<2J[b]	<2J[b]	<2[b]	<2J[b]	<2[b]	<50J[b]	<100J[b]	<100J[b]	<50J	<2J
RRPH (Approx.)	10-14	100-140	2,000[a]	<100-<600	<140	<120	<140	<120	<120	<100	NA	<2,000	NA	<2,000	<100
BTEX (8020/ 8020 Mod.)			10 Total BTEX	<0.10-<0.5	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1					
Benzene	0.002	0.02	0.5	<0.02-<0.1	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<1	<1	<1	<1	<0.02
Toluene	0.002	0.02		<0.02-<0.1	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<1	<1	<1	<1	<0.02
Ethylbenzene	0.002	0.02		<0.02-<0.1	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<1	<1	<1	<1	<0.02
Xylenes (Total)	0.004	0.04		<0.04-<0.2	<0.04	<0.04	<0.04	<0.04	<0.04	<0.04	<2	<2	<2	<2	<0.04
HVOC 8010	0.002	0.02		<0.02-<0.5	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<1	<10	<10	NA	<0.02
VOC 8260	0.020	0.02-0.03		<0.020-<0.400	NA	<0.030	NA	NA	<0.020	NA	NA	<1	<1	<1	<0.020
SVOC 8270															
di-n-Buthyl-phthalate	0.200	0.220-5.70	8,000	1.69U-83.4J	NA	8.08B	NA	NA	1.60B	NA	NA	<10	NA	<10	1.41
PCBs	0.01	0.1	10	<0.1-<2.8J	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	NA	<2J	NA	<2	<0.1

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
B Analyte was found in the associated blank.
J Result is an estimate.
U Compound is not present above the concentration listed.
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-3. DRUM STORAGE AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright Matrix: Soil

Site: Drum Storage Area (ST02) Units: mg/kg

METALS ANALYSES

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples						Field Blank			Lab Blank
					S02	S04						EB01		
Laboratory Sample ID Numbers					4479-4	4479-3						4479-7		4479
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg						µg/L		µg/L
Aluminum	0.35	2		1,500-25,000	9,600	2,600						<100		<100
Antimony	N/A	51-61		<7.8-<230	<61	<51						<100		<100
Arsenic	0.11	6.1-51		<4.9-8.5	<6.1	<51						<100		<100
Barium	0.24	1		27-390	150	180						<50		<50
Beryllium	N/A	26-31		<2.6-6.4	<31	<26						<50		<50
Cadmium	0.33	26-31		<3.0-<36	<31	<26						<50		<50
Calcium	0.69	4		360-59,000	910	15,000						410		327
Chromium	0.066	26-31		<4.3-47	<31	<26						<50		<50
Cobalt	N/A	1		<5.1-12	<6.1	15						<100		<100
Copper	0.045	1		<2.7-45	8.5	9.3						<50		<50
Iron	0.50	2		5,400-35,000	27,000	110,000						<100		<100
Lead	0.13	2		<5.1-22	<61	19						<100		<100
Magnesium	0.96	4		360-7,400	1,300	5,300						<200		<200
Manganese	0.025	1		25-290	170	1,400						<50		<50
Molybdenum	N/A	3.1-26		<2.5-<11	<3.1	<26						<50		<50
Nickel	0.11	1		4.2-46	12	24						<50		<50
Potassium	23	100		<300-2,200	930	590						<5,000		<5,000

☐ CT&E Data.
N/A Not analyzed.

TABLE G-3. DRUM STORAGE AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright   Matrix: Soil

Site: Drum Storage Area (ST02)   Units: mg/kg

METALS ANALYSES

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples						Field Blank			Lab Blank
					S02	S04						EB01		
Laboratory Sample ID Numbers					4479-4	4479-3						4479-7		4479
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg						µg/L		µg/L
Selenium	1.2	61-510		<7.8-<170	<61	<510						<100		<100
Silver	0.53	26-31		<3-<110	<31	<26						<50R		<50
Sodium	0.55	5		<160-680	170	840						410		<250
Thallium	0.011	0.25-0.30		<0.2-<1.2	<0.30	<0.25						<5		<5
Vanadium	0.036	1		6.3-59	35	39						<50		<50
Zinc	0.16	1		9.2-95	25	66						<50		<50

☐ CT&E Data.

R Result has been rejected.

TABLE G-4. DIESEL FUEL SPILLS ANALYTICAL DATA SUMMARY

Installation: Wainwright
Site: Diesel Fuel Spills (SS04)

Matrix: Soil
Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples						Field Blanks			Lab Blanks	
					S01 & S05 (Replicates)		S02-1	S03-1	S04-1	S06-1.5	AB01	EB02	TB02		
Laboratory Sample ID Numbers					1478 4483-2	1486 4483-3	1480	1482	1484	1488	1424	1498/1500 4483-9	1422 4482-6	#5-9193 #1&2-9293	#5-9193 #3&4-9293
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	5-10	50-100	500[a]	<50[b]-<300J[b]	4,800J[b]	4,900J[b]	<50[b]	<50[b]	<100[b]	<70[b]	NA	<1,000[b]	NA	<1,000J	<200
GRPH	0.2	2	100	<2J[b]-<5J[b]	120NJ[b]	120NJ[b]	<2J[b]	<2J[b]	14NJ[b]	<2J[b]	<50J[b]	<50J[b]	<50J[b]	<50J	<1J-<20
RRPH (Approx.)	10-15	100-150	2,000[a]	<100-<600	<110	<110	<110	<100	<150	<140	NA	<2,000	NA	<2,000	<2,000
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1-<0.5	23J	24J	<1.0J	<1.0J	<1.0J	<1.0J					
Benzene	0.02	0.2	0.5	<0.02-<0.1	<0.2J	<0.2J	<0.2J	<0.2J	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2
Toluene	0.02	0.2		<0.02-<0.1	<0.2J	<0.2J	<0.2J	<0.2J	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2
Ethylbenzene	0.02	0.2		<0.02-<0.1	6NJ	7NJ	<0.2J	<0.2J	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2
Xylenes (Total)	0.04	0.4		<0.04-<0.2	17NJ	17NJ	<0.4J	<0.4J	<0.4J	<0.4J	<2	<2	<2	<2	<0.04-<0.4
VOC 8260															
p-Isopropyl-toluene	0.020	0.200		<0.020-<0.400	0.237	<0.2	NA	NA	NA	NA	NA	NA	<1	<1	<0.020
Naphthalene	0.020	0.200		<0.020-<0.400	0.613	0.851	NA	NA	NA	NA	NA	NA	<1	<1	<0.020
1,2,4-Trimethyl-benzene	0.020	0.200		<0.020-<0.400	0.382	0.849	NA	NA	NA	NA	NA	NA	<1	<1	<0.020
1,3,5-Trimethyl-benzene	0.020	0.200		<0.020-<0.400	5.49	14.4	NA	NA	NA	NA	NA	NA	<1	<1	<0.020
SVOC 8270	0.200	2.20		<0.200-83.4J	NA	<2.20	NA	NA	NA	NA	NA	NA	NA	NA	<0.200
Pesticides	0.001-0.05	0.01-0.5		<0.01J-<2.8J	NA	NA	<0.01J-<0.5J	NA	<0.01J-<0.5J	NA	NA	<0.02J-<10J	NA	NA	<0.01J-<0.5J
PCBs	0.01-0.04	0.1-0.4	10	<0.1-<2.8J	<0.1	<0.1	<0.1	<0.4	<0.1	<0.1	NA	<2	NA	<2	<2

☐ CT&E Data.
NA Not analyzed.
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-4. DIESEL FUEL SPILLS ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Diesel Fuel Spills (SS04)
Matrix: Soil/Sediment
Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks			Lab Blanks	
					SD01-1	SD02-1	SD03	SD04-1	AB01	EB02	TB02		
Laboratory Sample ID Numbers					1440 4483-1	1434	1436	1438	1424	1498/1500 4483-9	1422 4482-6	#5-9193 #1&2-9293 4482/4483	#6-91093 #3&4-9493 4483
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	5-33	50-330	500[a]	<50[b]-<300J[b]	<240[b]	<330[b]	<100[b]	<50[b]	NA	<200[b]	NA	<1,000J	<50
GRPH	0.2	2	100	<2J[b]-<5J[b]	<2J[b]	<2J[b]	6NJ[b]	<2J[b]	<50J[b]	<50[b]	<50J[b]	<50J	<1-<20
RRPH (Approx.)	11-67	110-670	2,000[a]	<100-<600	<480	<670	<200	<110	NA	<2,000	NA	<2,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1-<0.5	<1.0J	<1.0J	<1.0J	<1.0J					
Benzene	0.02	0.2	0.5	<0.02-<0.1	<0.2J	<0.2J	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2
Toluene	0.02	0.2		<0.02-<0.1	<0.2J	<0.2J	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2
Ethylbenzene	0.02	0.2		<0.02-<0.1	<0.2J	<0.2J	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2
Xylenes (Total)	0.04	0.4		<0.04-<0.2	<0.4J	<0.4J	<0.4J	<0.4J	<2	<2	<2	<2	<0.04-<0.4
VOC 8260	0.020	0.075		<0.020-<0.400	<0.075	NA	NA	NA	NA	NA	<1	<1	<0.020

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
N The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-4. DIESEL FUEL SPILLS ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Diesel Fuel Spills (SS04)
Matrix: Soil
Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples		Field Blanks			Lab Blanks	
					2S07	2S08-1	AB01	2EB03	2TB03		
Laboratory Sample ID Numbers					1877	1878 4695-1	1424	1894/1896	1886	#6-9993 #1&2-9793 #1&2-9293	#6-91093 #1&2-91093 4695
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	4.00-6	4.00-60	500[a]	<50[b]-<300[b]	<60J[b]	2,220[c]	NA	<1,000J[b]	NA	<1,000	<4.00-<50
GRPH	0.400	0.400	100	<2J[b]<5J[b]	NA	46.2	<50J[b]	<50J[b]	<50J[b]	<2J-<50J	<0.400
RRPH (Approx.)	14-16	140-160	2,000[a]	<100-<600	<140	<160	NA	<2,000	NA	<2,000	<100

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.
c This sample was analyzed by F&B also; DRPH were detected at 750J[b] mg/kg.

TABLE G-4. DIESEL FUEL SPILLS ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright — Matrix: Surface Water
Site: Diesel Fuel Spill (SS04) — Units: µg/L

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples: SW01 & SW04 (Duplicates)		Environmental Samples: SW02	Environmental Samples: SW03	Field Blanks: AB01	Field Blanks: EB02	Field Blanks: TB02	Lab Blanks
Laboratory Sample ID Numbers					1406/1408 4482-1	1417/1418 4482-5	1426/1428	1430/1432	1424	1498/1500 4483-9	1422 4482-6	4482 4483 #5-9193 #1&2-9293
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
DRPH	100	1,000		<1,000J[b]	<1,000[b]	<1,000[b]	<1,000[b]	<1,000[b]	NA	<200[b]	NA	<1,000J
GRPH	5	50		<50J[b]-<100J[b]	<50J[b]	<50R[b]	<50J[b]	<50J[b]	<50J[b]	<50J[b]	<50J[b]	<50J
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000	<2,000	<2,000	NA	<2,000	NA	<2,000
BTEX (8020/8020 Mod.)												
Benzene	0.1	1	5	<1	<1	<1R	<1	<1	<1	<1	<1	<1
Toluene	0.1	1	1,000	<1	<1	<1R	<1	<1	<1	<1	<1	<1
Ethylbenzene	0.1	1	700	<1	<1	<1R	<1	<1	<1	<1	<1	<1
Xylenes (Total)	0.2	2	10,000	<2	<2	<2R	<2	<2	<2	<2	<2	<2
HVOC	0.1	1		<5-<10	<1	NA	NA	NA	<1	<1	<1	<1
VOC 8260	1	1		<1	<1	<1	NA	NA	NA	NA	<1	<1
SVOC 8270	10	10		<10-<13	<10	<10	NA	NA	NA	NA	NA	<10
TOC	5,000	5,000		7,480	32,500	30,000	NA	NA	NA	<5,000	NA	<5,000
TSS	100	200		7,000-35,000	28,000	13,000	NA	NA	NA	NA	NA	<100
TDS	10,000	10,000		91,000-151,000	459,000	465,000	NA	NA	NA	NA	NA	<10,000

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
R Result has been rejected.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-5. LANDFILL ANALYTICAL DATA SUMMARY

Installation: Wainwright
Site: Landfill (LF05)
Matrix: Soil
Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks			Lab Blanks	
					S01-1	S02-1	S03-1 & S07-1 (Replicates)		S04-1.5	AB01	EB01	TB01		
Laboratory Sample ID Numbers					1222	1224	1226	1230	1228 4479-2	1424	1280/1282 4479-7	1260 4479-8	#5-9193 #1&2-9293 4479	#6-9393 #6-83193 #3&4-9293 4479
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	7-12	70-120	500[a]	<50[b]-<300J[b]	<110[b]	<120[b]	<70[b]	<100[b]	60[b]	NA	<1,000[b]	NA	<1,000J	<50
GRPH	0.2-0.4	2-4	100	<2J[b]-<5J[b]	<4[b]	<4[b]	<2J[b]	<2J[b]	200J[b]	<50J[b]	<100J[b]	<100J[b]	<50J	<2J
RRPH (Approx.)	12-24	120-240	2,000[a]	<100-<600	<220	<240	<140	<140	<120	NA	<2,000	NA	<2,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1-<0.5	<0.20	<0.20	<0.10	<0.10J	9.59J					
Benzene	0.002-0.004	0.02-0.04	0.5	<0.02-<0.1	<0.04	<0.04	<0.02	<0.02J	<0.02	<1	<1	<1	<1	<0.02
Toluene	0.002-0.004	0.02-0.04		<0.02-<0.1	<0.04	<0.04	<0.02	<0.02	0.08J	<1	<1	<1	<1	<0.02
Ethylbenzene	0.002-0.004	0.02-0.04		<0.02-<0.1	<0.04	<0.04	<0.02	<0.02	1.5J	<1	<1	<1	<1	<0.02
Xylenes (Total)	0.004-0.008	0.04-0.08		<0.04-<0.2	<0.08	<0.08	<0.04	<0.04	8J	<2	<2	<2	<2	<0.04
HVOC 8010	0.002-0.004	0.02-0.04		<0.02-<0.5	<0.04	<0.04	<0.02	<0.02	<0.02	<10	<10	<10	<10	<0.02
VOC 8260														
Toluene	0.020	0.2-0.3		<0.020-<0.400	NA	NA	NA	NA	0.205	NA	<1	<1	<1	<0.020
1,3,5-Trimethylbenzene	0.020	0.2-0.3		<0.020-<0.400	NA	NA	NA	NA	0.247	NA	<1	<1	<1	<0.020
Xylenes (Total)	0.040	0.4-0.6		<0.020-<0.400	NA	NA	NA	NA	0.211	NA	<1	<1	<2	<0.040
SVOC 8270	0.20	0.21		1.69U-83.4J	NA	NA	NA	NA	<0.21-3.41U	NA	<10	NA	<10	1.41

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate
U Compound is not present above the concentration listed.
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-5. LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Landfill (LF05)

Matrix: Soil
Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks			Lab Blanks	
					S01-1	S02-1	S03-1 & S07-1 (Replicates)		S04-1.5	AB01	EB01	TB01		
Laboratory Sample ID Numbers					1222	1224	1226	1230	1228 4479-2	1424	1280/1282 4479-7	1260 4479-8	#5-9193 4479	#6-9393 #6-83193 4479
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
Pesticides	0.001-0.05	0.01-0.5		<0.01J-<2.8J	NA	NA	NA	NA	<0.01J-0.5J	NA	<0.2J-<10J	NA	NA	<0.5
PCBs	0.01	0.1	10	<0.1-<2.8J	<0.1	<0.1	<0.1	<0.1	<0.1	NA	<2J	NA	<2	<0.1
TOC				10,500-44,100	NA	NA	NA	NA	25,900	NA	<5,000	NA	<5,000	NA

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.

TABLE G-5. LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Landfill (LF05)

Matrix: Sediment
Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples		Field Blanks			Lab Blanks	
					SD01	SD02	AB01	EB01	TB01		
Laboratory Sample ID Numbers					1232	1234 4479-1	1424	1280/1284 4479-7	1260 4479-8	#5-9193 #1&2-9293	#6-83193 #3&4-9293 4479
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	13-49	130-490	500[a]	<50[b]-<300J[b]	<490[b]	<130[b]	NA	<1,000[b]	NA	<1,000J	<50
GRPH	0.6-0.8	6-8	100	<2J[b]-<5J[b]	<8J[b]	<6J[b]	<50J[b]	<100J[b]	<100J[b]	<50J	<2J
RRPH (Approx.)	26-43	260-430	2,000[a]	<100-<600	<430	<260	NA	<2,000	NA	<2,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1-<0.5	<0.45J	<0.25J					
Benzene	0.005-0.009	0.05-0.09	0.5	<0.02-<0.1	<0.09J	<0.05J	<1	<1	<1	<1	<0.02
Toluene	0.005-0.009	0.05-0.09		<0.02-<0.1	<0.09	<0.05	<1	<1	<1	<1	<0.02
Ethylbenzene	0.005-0.009	0.05-0.09		<0.02-<0.1	<0.09	<0.05	<1	<1	<1	<1	<0.02
Xylenes (Total)	0.01-0.018	0.1-0.18		<0.04-<0.2	<0.18	<0.1	<2	<2	<2	<2	<0.04
HVOC 8010	0.002	0.02		<0.02-<0.5	<0.02	<0.02	<1	<10	<10	NA	<0.02
VOC 8260	0.020	0.3		<0.020-<0.400	NA	<0.3	NA	<1	<1	<1	<0.020
SVOC 8270											
di-n-Butylphthalate	0.200	3.2	8,000	1.69U-83.4J	NA	37.6J	NA	<10	NA	<10	1.41
PCBs	0.03-0.04	0.3-0.4	10	<0.1J-<2.8J	<0.4	<0.3	NA	<2J	NA	<2	<0.1
TOC				10,500-44,100	NA	27,100	NA	<5,000	NA	<5,000	NA

☐ CT&E Data.
░ F&B Data.
NA Not analyzed.
J Result is an estimate.
U Compound is not present above the concentration listed.
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-5. LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright Matrix: Soil/Sediment

Site: Landfill (LF05) Units: mg/kg

METALS ANALYSES

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples					Field Blank			Lab Blank
					S04-1.5	SD02					EB01		
Laboratory Sample ID Numbers					4479-2	4479-1					4479-7		4479
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg					µg/L		µg/L
Aluminum	0.35	2		1,500-25,000	1,900	15,000					<100		<100
Antimony	N/A	51-150		<7.8-<230	<51J	<150J					<100		<100
Arsenic	0.11	51-150		<4.9-8.5	<51	<150					<100		<100
Barium	0.024	1		27-390	250	420					<50		<50
Beryllium	N/A	7.2-26		<2.6-64	<26	<7.2					<50		<50
Cadmium	0.33	1-26		<3.0-<36	<26	72					<50		<50
Calcium	0.69	4		360-59,000	2,700	4,950					410		327
Chromium	0.066	1-26		<4.3-47	<26	26					<50		<50
Cobalt	N/A	1-15		<5.1-12	12	<15					<100		<100
Copper	0.045	1		<2.7-45	41	14					<50		<50
Iron	0.50	2		5,400-35,000	99,000	25,000					<100		<100
Lead	0.13	2-15		<5.1-22	37	<15					<100		<100
Magnesium	0.96	4		360-7,400	1,900	3,100					<200		<200
Manganese	0.025	1		25-290	1,100J	67J					<50		<50
Molybdenum	N/A	7.2-26		<2.5-<11	<26	<7.2					<50		<50
Nickel	0.11	1		4.2-46	25	15					<50		<50
Potassium	23	100		<300-2,200	510J	1,500J					<5,000		<5,000

☐ CT&E Data.
N/A Not available.
J Result is an estimate

TABLE G-5. LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright | Matrix: Soil/Sediment | METALS ANALYSES

Site: Landfill (LF05) | Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples					Field Blank			Lab Blank
					S04-1.5	SD02					EB01		
Laboratory Sample ID Numbers					4479-2	4479-1					4479-7		4479
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg					µg/L		µg/L
Selenium	1.2	150-510		<7.8-<170	<510	<150					<100		<100
Silver	0.53	26-72		<3-<110	<26R	<72R					<50R		<50
Sodium	0.55	5		<160-680	84	1,500					410		<250
Thallium	0.011	0.26-0.82		<0.2-<1.2	<0.26	<0.82					<5		<5
Vanadium	0.036	1		6.3-59	30	43					<50		<50
Zinc	0.16	1		9.2-95	150	30					<50		<50

☐ CT&E Data.

R Result has been rejected.

TABLE G-5. LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright Matrix: Surface Water
Site: Landfill (LF05) Units: µg/L

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples SW01	Environmental Samples SW02			Field Blanks AB01	Field Blanks EB01	Field Blanks TB01	Lab Blanks
Laboratory Sample ID Numbers					1255/1256 1258 4478-1	1250/1252 4478-2			1424	1280/1282 4479-7	1260 4479-8	#5-9193 #1&2-9293 4478/4479
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L			µg/L	µg/L	µg/L	µg/L
DRPH	100	1,000		<1,000J[b]	<1,000[b]	<1,000[b]			NA	<1,000[b]	NA	<1,000J
GRPH	10	100		<50J[b]-<100J[b]	<100J[b]	<100J[b]			<50J[b]	<100J[b]	<100J[b]	<50J
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000			NA	<2,000	NA	<2,000
BTEX (8020/8020 Mod.)												
Benzene	0.1	1	5	<1	<1	<1			<1	<1	<1	<1
Toluene	0.1	1	1,000	<1	<1	<1			<1	<1	<1	<1
Ethylbenzene	0.1	1	700	<1	<1	<1			<1	<1	<1	<1
Xylenes (Total)	0.2	2	10,000	<2	<2	<2			<2	<2	<2	<2
HVOC 8010	1	10		<5-<10	<10	<10			<1	<10	<10	NA
VOC 8260												
1,2-Dichloroethane	1	1	5	<1	<1	6.2			NA	<1	<1	<1
SVOC 8270	10	10-12		<10-<13	<10	<12			NA	<10	NA	<10
PCBs	0.2	2	0.5	<2J	<2	<2			NA	<2J	NA	<2
TOC	5,000	5,000		7,480	231,000	64,600			NA	<5,000	NA	<5,000
TSS	100	200		7,000-35,000	105,000	68,000			NA	NA	NA	<100
TDS	10,000	10,000		91,000-151,000	1,060,000J	90,000			NA	NA	NA	<10,000

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-5. LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright Matrix: Surface Water

Site: Landfill (LF05) Units: µg/L

METALS ANALYSES: TOTAL
(DISSOLVED)

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples					Field Blank			Lab Blanks
					SW01	SW02					EB01		
Laboratory Sample ID Numbers					4478-1	4478-2					4479-7		4478 4479
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L					µg/L		µg/L
Aluminum	17.4	100		<100-350 (<100-340)	2,100 (2,200)	180 (<100)					<100		<100
Antimony	N/A	100	6	<100 (<100)	<100 (<100)	<100 (<100)					<100		<100
Arsenic	5.3	100	50	<100 (<100)	<100 (<100)	<100 (<100)					<100		<100
Barium	1.2	50	2,000	<50-93 (50-91)	230 (230)	53 (<50)					<50		<50
Beryllium	N/A	50	4	<50 (<50)	<50 (<50)	<50 (<50)					<50		<50
Cadmium	1.7	50	5	<50 (<50)	<50 (<50)	<50 (<50)					<50		<50
Calcium	34.5	200		4,500-88,000 (4,100-86,000)	24,000 (23,000)	6,000 (5,200)					410		<200
Chromium	3.29	50	100	<50 (<50)	<50 (<50)	<50 (<50)					<50		<50
Cobalt	N/A	100		<100 (<100)	<100 (<100)	<100 (<100)					<100		<100
Copper	2.3	50	1,300	<50 (<50)	<50 (<50)	<50 (<50)					<50		<50
Iron	25	100		180-2,800 (<100-1,600)	23,000 (21,000)	1,600 (480)					<100		<100

☐ CT&E Data.
N/A Not available.

TABLE G-5. LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright — Matrix: Surface Water

Site: Landfill (LF05) — Units: μg/L

METALS ANALYSES: TOTAL (DISSOLVED)

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples					Field Blank			Lab Blanks
					SW01	SW02					EB01		
Laboratory Sample ID Numbers					4478-1	4478-2					4479-7		4478 4479
ANALYSES	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L					μg/L		μg/L
Lead	6.6	100	15	<100 (<100)	<100 (<100)	<100 (<100)					<100		<100
Magnesium	47.8	200		<5,000-53,000 (2,600-54,000)	26,000 (27,000)	6,000 (5,000)					<200		<200
Manganese	1.24	50		<50-510 (<50-120)	150 (130)	<50 (<50)					<50		<50
Molybdenum	N/A	50		<50 (<50)	<50 (<50)	<50 (<50)					<50		<50
Nickel	5.5	50	100	<50 (<50)	<50 (<50)	<50 (<50)					<50		<50
Potassium	1,154	5,000		<5,000 (<5,000)	9,500 (11,000)	<5,000 (<5,000)					<5,000		<5,000
Selenium	62.4	100	50	<100 (<100)	<100 (<100)	<100 (<100)					<100		<100
Silver	2.6	50	50	<50 (<50)	<50 (<50)J	<50 (<50)					<50R		<50
Sodium	27.7	200		8,400-410,000 (8,200-450,000)	110,000 (130,000)	18,000 (10,000)					410		410
Thallium	0.57	5	2	<5 (<5)	<5 (<5)	<5 (<5)					<5		<5

☐ CT&E Data.
N/A Not available.
J Result is an estimate.
R Result has been rejected.

TABLE G-5. LANDFILL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright   Matrix: Surface Water

Site: Landfill (LF05)   Units: µg/L

METALS ANALYSES: TOTAL (DISSOLVED)

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples					Field Blank			Lab Blanks
					SW01	SW02					EB01		
Laboratory Sample ID Numbers					4478-1	4478-2					4479-7		4478 4479
ANALYSES	µg/L	µg/L	. µg/L	µg/L	µg/L	µg/L					µg/L		µg/L
Vanadium	1.8	50		<50 (<50)	<50 (<50)	<50 (<50)					<50		<50
Zinc	8.2	50		<50-160 (<50)	230J (160)	<50 (<50)					<50		<50

☐ CT&E Data.
N/A Not available.
J Result is an estimate.
R Result has been rejected.

TABLE G-6. GARAGE ANALYTICAL DATA SUMMARY

Installation: Wainwright   Matrix: Soil/Sediment
Site: Garage (SS07)   Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples: S01 & S03 (Replicates)		Environmental Samples: S02	Environmental Samples: SD01	Environmental Samples: SD02	Environmental Samples: SD03	Environmental Samples: SD04	Field Blanks: AB01	Field Blanks: EB02	Field Blanks: TB02	Lab Blanks	
Laboratory Sample ID Numbers					1458 4484-3	1462 4484-4	1460	1452 4484-5	1454	1456	1450	1424	1498 1500	1422 4482-6	#5-9193 #1&2-9293 4482	#5-9193 #3&4-9293 #3&4-9493 4484
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	5-7	50-70	500[a]	<50[b]-<300J[b]	6,500J[b]	8,300J[b]	570J[b]	<50[b]	<70[b]	<50[b]	<60[b]	NA	<1,000[b]	NA	<1,000J	<200
GRPH	0.2-2	2-20	100	<2J[b]-<5J[b]	120NJ[b]	59NJ[b]	8NJ[b]	<2J[b]	<2J[b]	<20J[b]	<2J[b]	<50J[b]	<50J[b]	<50J[b]	<50J	<1J-<20
RRPH (Approx.)	10-14	100-140	2,000[a]	<100-<600	1,200	2,400	530	<100	<140	<110	<120	NA	<2,000	NA	<2,000	<2,000
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.10-<0.5	19J	1.9J	1.2J	<1.0J	0.6J	<1.0J	<1.0J					
Benzene	0.002-0.02	0.02-0.2	0.5	<0.02-<0.1	<0.2J	<0.2	<0.2J	<0.2J	<0.02J	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2
Toluene	0.002-0.02	0.02-0.2		<0.02-<0.1	<0.2J	<0.2	<0.2J	<0.2J	<0.02J	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2
Ethyl-benzene	0.002-0.02	0.02-0.2		<0.02-<0.1	4NJ	0.7NJ	<0.2J	<0.2J	0.1JN	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2
Xylenes (Total)	0.004-0.04	0.04-0.4		<0.04-<0.2	15NJ	1.2NJ	1.2NJ	<0.4J	0.5JN	<0.4J	<0.4	<2	<2	<2	<2	<0.04-<0.4
HVOC 8010																
Tetrachloro-ethene	0.002	0.02		<0.02-<0.5	10	7	2	<0.02	<0.02	<0.02	<0.02	<1	<1	<1	<1	<0.02
VOC 8260																
p-Iso-propyl-toluene	0.020	0.020-0.200		<0.020-<0.400	0.394	0.502	NA	<0.020	NA	NA	NA	NA	NA	<1	<1	<0.020
Naph-thalene	0.020	0.020-0.200		<0.020-<0.400	<0.200	0.393	NA	0.034	NA	NA	NA	NA	NA	<1	<1	<0.020

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
N The analysis indicates the presence of an analyte for which there is presumptive evidence to make a "tentative identification".
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-6. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright Matrix: Soil/Sediment
Site: Garage (SS07) Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples: S01 & S03 (Replicates)		S02	SD01	SD02	SD03	SD04	Field Blanks: AB01	EB02	TB02	Lab Blanks	
Laboratory Sample ID Numbers					1458 4484-3	1462 4484-4	1460	1452 4484-5	1454	1456	1450	1424	1498 1500	1422 4482-8	#5-9193 4482	#5-9193 4484
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
Tetra-chloro-ethene	0.020	0.020-0.200		<0.020-<0.400	10.4	11.5	NA	0.059	NA	NA	NA	NA	NA	<1	<1	<0.020
1,2,4-Tri-methyl-benzene	0.020	0.020-0.200		<0.020-<0.400	0.376	0.714	NA	0.040	NA	NA	NA	NA	NA	<1	<1	<0.020
1,3,5-Tri-methyl-benzene	0.020	0.020-0.200		<0.020-<0.400	1.75	5.36	NA	0.024	NA	NA	NA	NA	NA	<1	<1	<0.020
Xylenes (Total)	0.040	0.040-0.400		<0.040-<0.800	<0.400	0.354	NA	0.022	NA	NA	NA	NA	NA	<2	<2	<0.040
Pesticides	0.001-0.05	0.01-0.5		<0.01J-<2.8J	<0.01J-<0.5J	NA	NA	<0.01J-<0.5J	<0.01J-<0.5J	NA	NA	NA	<0.2J-<10J	NA	NA	<0.01-<0.5J
PCBs	0.01-0.20	0.1-2	10	<0.1-<2.8J	<2	<2J	<2	<0.1	<0.1	<0.1	<0.1	<2	<2	NA	<2	<2

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.

TABLE G-6. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright   Matrix: Sediment
Site: Garage (SS07)   Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks			Lab Blanks	
					2SD05	2SD06-1			AB01	2EB03	2TB03		
Laboratory Sample ID Numbers					1891	1892 4695-2			1424	1894/1896 4695-3	1886 4694-9	#6-9993 #1&2-9793 #1&2-9293	#6-91093 4695
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg			µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	4.00-8	4.00-80	500[a]	<50[b]-<300J[b]	120,000J[b]	47.4[cd]			NA	<1,000J[b]	NA	<1,000	<4.00-<50
GRPH	0.400	0.600	100	<2J[b]-<5J[b]	NA	<0.600			<50J[b]	<50J[b]	<50J[b]	<2J-<50J	<0.400
RRPH (Approx.)	16	160	2,000[a]	<100-<600	77,000	<160			NA	<2,000	NA	<2,000	<100

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.
c This sample was analyzed by F&B also; DRPH were detected at <80J[b] mg/kg.
d The laboratory reported that the EPH pattern in this sample was not consistent with a middle distillate fuel.

TABLE G-6. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Garage (SS07)

Matrix: Soil/Sediment
Units: mg/kg

METALS ANALYSES

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples						Field Blank			Lab Blanks
					S01 & S03 (Replicates)		SD01					EB02		
Laboratory Sample ID Numbers					4484-3	4484-4	4484-5					4483-9		4483 4484
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg					µg/L		µg/L
Aluminum	0.35	2-3,600		1,500-25,000	<3,600	2,800	1,980					<100		<100
Antimony	N/A	50-54		<7.8-<230	<53	<54	<50R					<100		<100
Arsenic	0.11	50-54		<4.9-8.5	<53	<54	<50					<100		<100
Barium	0.024	1		27-390	160	240	220					<50		<50
Beryllium	N/A	25-27		<2.6-6.4	<27	<27	<25					<50		<50
Cadmium	0.33	25-27		<3.0-<36	<27	<27	<25					<50		<50
Calcium	0.69	4		360-59,000	5,200	6,100	3,700					<200		<200
Chromium	0.066	1-27		<4.3-47	30	<27	<25					<50		<50
Cobalt	N/A	1-13.5		<5.1-12	8.6	<5.4	<13.5					<100		<100
Copper	0.045	1		<2.7-45	39	13	17					<50		<50
Iron	0.50	2		5,400-35,000	53,000	36,000	114,000					<100		<100
Lead	0.13	2-50		<5.1-22	130	74	<50					<100		<100
Magnesium	0.96	4		360-7,400	3,300	2,960	2,500					<200		<200
Manganese	0.025	1		25-290	660	370	1,250					<50		<50
Molybdenum	N/A	2.7-25		<2.5-<11	<2.7	<2.7	<25					<50		<50
Nickel	0.11	1		4.2-46	18	14	29					<50		<50
Potassium	23	100-270		<300-2,200	425	<270	290					<5,000		<5,000

☐ CT&E Data.
N/A Not available.
R Result has been rejected.

TABLE G-6. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Garage (SS07)

Matrix: Soil/Sediment
Units: mg/kg

METALS ANALYSES

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples						Field Blank			Lab Blanks
					S01 & S03 (Replicates)		SD01					EB02		
Laboratory Sample ID Numbers					4484-3	4484-4	4484-5					4483-9		4483 4484
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg					µg/L		µg/L
Selenium	1.2	53-500		<7.8-<170	<53	<54	<500					<100		<100
Silver	0.53	25-27		<3-<110	<27	<27	<25R					<50J		<50
Sodium	0.55	5		<160-680	100	95	100					<250		<250
Thallium	0.011	0.26-0.27		<0.2-<1.2	<0.27	<0.27	<0.26					<5		<5
Vanadium	0.036	1		6.3-59	21	16	34					<50		<50
Zinc	0.16	1		9.2-95	240	89	160					<50		<50

☐ CT&E Data.
J Result is an estimate.
R Result has been rejected.

TABLE G-6. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright   Matrix: Surface Water
Site: Garage (SS07)   Units: µg/L

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks			Lab Blanks
					SW01	SW02	SW03			AB01	EB02	TB02	
Laboratory Sample ID Numbers					1464/1466 4484-1	1468/1470	1474 4484-2			1424	1498/1500	1422 4482-6	#5-9193 #1&2-8293 4484/4482/4483
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L			µg/L	µg/L	µg/L	µg/L
DRPH	100	1,000		<1,000J[b]	<1,000J[b]	<1,000[b]	<1,000[b]			NA	<1,000[b]	NA	<1,000J
GRPH	5	50		<50J[b]-<100J[b]	<50J[b]	<50J[b]	<50J[b]			<50J[b]	<50J[b]	<50J[b]	<2J-<50J
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000	<2,000			NA	<2,000	NA	<2,000
BTEX (8020/ 8020 Mod.)													
Benzene	0.1	1	5	<1	<1	<1	<1			<1	<1	<1	<1
Toluene	0.1	1	1,000	<1	<1	<1	<1			<1	<1	<1	<1
Ethylbenzene	0.1	1	700	<1	<1	<1	<1			<1	<1	<1	<1
Xylenes (Total)	0.2	2	10,000	<2	<2	<2	<2			<2	<2	<2	<2
VOC 8260													
1,2-Dichloroethane	1	1	5	<1	1.8J	NA	<1			NA	NA	<1	<1
SVOC 8270													
bis-(2-Ethylhexyl) phthalate	10	10-11		<10-<13	<11	NA	16			NA	NA	NA	<10
Pesticides	0.02-1	0.2-10		<0.2J-<25J	<0.2J-<10J	<0.2J-<10J	NA			NA	<0.2J-<10J	NA	<0.01-<0.5J

□ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-6. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright — Site: Garage (SS07)

Matrix: Surface Water — Units: µg/L

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blank			Lab Blanks
					2SW04 & 2SW06 (Replicates)		2SW05				2EB03		
Laboratory Sample ID Numbers					1888	1890	1889				1894 1896		#6-9993 #5-9193
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L				µg/L		µg/L
DRPH	100	1,000		<1,000J[b]	<1,000[b]	<1,000[b]	<1,000[b]				<1,000J[b]		<1,000J
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000	<2,000				<2,000		<2,000

☐ CT&E Data.
▩ F&B Data.
J Result is an estimate.
b DRPH concentrations reported for these samples are equivalent to diesel range organics (DRO) as defined by ADEC.

TABLE G-6. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Garage (SS07)

Matrix: Surface Water
Units: μg/L

METALS ANALYSES: TOTAL
(DISSOLVED)

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range for 7 DEW Line Installations	Environmental Samples						Field Blank			Lab Blanks
					SW01	SW03						EB02		
Laboratory Sample ID Numbers					4484-1	4484-2						4483-9		4483 4484
ANALYSES	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L						μg/L		μg/L
Aluminum	17.4	100		<100-350 (<100-340)	<100 (<100)	<100 (<100)						<100		<100
Antimony	N/A	100	6	<100 (<100)	<100 (<100)	<100 (<100)						<100		<100
Arsenic	5.3	100	50	<100 (<100)	<100 (<100)	<100 (<100)						<100		<100
Barium	1.2	50	2,000	<50-93 (<50-91)	220 (180)	240 (200)						<50		<50
Beryllium	N/A	50	4	<50 (<50)	<50 (<50)	<50 (<50)						<50		<50
Cadmium	1.7	50	5	<50 (<50)	<50 (<50)	<50 (<50)						<50		<50
Calcium	34.5	200		4,500-8,800 (4,100-86,000)	30,000 (35,000)	37,000 (29,000)						<200		<200
Chromium	3.29	50	100	<50 (<50)	<50 (<50)	<50 (<50)						<50		<50
Cobalt	N/A	100		<100 (<100)	<100 (<100)	<100 (<100)						<100		<100
Copper	2.3	50	1,300	<50 (<50)	<50 (<50)	<50 (<50)						<50		<50
Iron	25	100		180-2,800 (<100-1,600)	2,900 (300)	1,100 (130)						<100		<100
Lead	6.6	100	15	<100 (<100)	<100 (<100)	<100 (<100)						<100		<100

☐ CT&E Data.
N/A Not available.

TABLE G-6. GARAGE ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Garage (SS07)

Matrix: Surface Water
Units: µg/L

METALS ANALYSES: TOTAL
(DISSOLVED)

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range for 7 DEW Line Installations	Environmental Samples						Field Blank			Lab Blanks
					SW01	SW03						EB02		
Laboratory Sample ID Numbers					4484-1	4484-2						4483-9		4483 4484
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L						µg/L		µg/L
Magnesium	47.8	200		<5,000-53,000 (2,600-54,000)	41,000 (60,000)	62,000 (41,000)						<200		<200
Manganese	1.24	50		<50-510 (<50-120)	<50 (<50)	<50 (<50)						<50		<50
Molybdenum	N/A	50		<50 (<50)	<50 (<50)	<50 (<50)						<50		<50
Nickel	5.5	50	100	<50 (<50)	<50 (<50)	<50 (<50)						<50		<50
Potassium	1,154	5,000		<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)						<5,000		<5,000
Selenium	62.4	100	50	<100 (<100)	<100 (<100)	<100 (<100)						<100		<100
Silver	2.6	50	50	<50 (<50)	<50 (<50)	<50 (<50)						<50J		<50
Sodium	27.7	250		8,400-410,000 (8,200-450,000)	49,000 (51,000)	51,000 (48,000)						<250		<250
Thallium	0.57	5	2	<5 (<5)	<5 (<5)	<5 (<5)						<5		<5
Vanadium	1.8	50		<50 (<50)	<50 (<50)	<50 (<50)						<50		<50
Zinc	8.2	50		<50-160 (<50)	440J (<50)	<50 (<50)						<50		<50

☐ CT&E Data.
N/A Not available.
J Result is an estimate

TABLE G-7. AIRSTRIP DIESEL ANALYTICAL DATA SUMMARY

Installation: Wainwright
Site: Airstrip Diesel (SS08)

Matrix: Sediment
Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples					Field Blanks			Lab Blanks	
					SD01	SD02	SD03	SD04 & SD05 (Replicates)		AB01	EB01	TB01		
Laboratory Sample ID Numbers					1240	1242 4480-1	1244	1246	1248	1424	1280/1282 4479-7	1260 4479-8	#5-9193 #1&2-9293 4479	#6-83193 #3&4-9293 4480
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	7.4-18	74-180	500[a]	<50[b]-<300J[b]	<81[b]	<74[b]	<135[b]	<120[b]	<180[b]	NA	<1,000[b]	NA	<1,000J	<50
GRPH	0.3-0.5	3-5	100	<2J[b]-<5J[b]	<3J[b]	<3J[b]	<5J[b]	<5J[b]	NA	<50J[b]	<100J[b]	<50[b]	<50J	<2J
RRPH (Approx.)	14-36	140-360	2,000[a]	<100-<600	<160	<140	<280	<250	<360	NA	<2,000	NA	<2,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1-<0.5	<0.15	<0.15	<0.25	<0.87J	NA					
Benzene	0.003-0.005	0.03-0.05	0.5	<0.02-<0.1	<0.03	<0.03	<0.05	<0.05	NA	<1	<1	<1	<1	<0.02
Toluene	0.003-0.05	0.03-0.5		<0.02-<0.1	<0.03	<0.03	<0.05	<0.5J	NA	<1	<1	<1	<1	<0.02
Ethylbenzene	0.003-0.005	0.03-0.05		<0.02-<0.1	<0.03	<0.03	<0.05	<0.04	NA	<1	<1	<1	<1	<0.02
Xylenes (Total)	0.006-0.001	0.06-0.1		<0.04-<0.2	<0.06	<0.06	<0.1	<0.08	NA	<2	<2	<2	<2	<0.04
VOC 8260	0.020	0.025		<0.020-<0.400	NA	<0.025	NA	NA	NA	NA	<1	<1	<1	<0.020

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-7. AIRSTRIP DIESEL ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Airstrip Diesel (SS08)

Matrix: Surface Water
Units: μg/L

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks			Lab Blanks
					SW01	SW02	SW03 & SW04 (Duplicates)		AB01	EB01	TB01	
Laboratory Sample ID Numbers					1267/1268	1262/1264 4480-2	1271/1272	1275/1276	1424	1280/1282 4479-7	1260 4479-8	#5-9193 #1&2-9293 4479/4480
ANALYSES	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L	μg/L
DRPH	100	1,000		<1,000J[b]	<1,000[b]	<1,000[b]	<1,000[b]	<1,000[b]	NA	<1,000[b]	NA	<1,000J
GRPH	10	100		<50J[b]-<100J[b]	<100J[b]	<100J[b]	<100J[b]	<100J[b]	<50J[b]	<100J[b]	<100J[b]	<50J
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000	<2,000	<2,000	NA	<2,000	NA	<2,000
BTEX (8020/8020 Mod.)												
Benzene	0.1	1	5	<1	<1	<1	<1	<1	<1	<1	<1	<1
Toluene	0.1	1	1,000	<1	<1	<1	<1	<1	<1	<1	<1	<1
Ethylbenzene	0.1	1	700	<1	<1	<1	<1	<1	<1	<1	<1	<1
Xylenes (Total)	0.2	2	10,000	<2	<2	<2	<2	<2	<2	<2	<2	<2
VOC 8260	1	1		<1	NA	<1	NA	NA	NA	<1	<1	<1
SVOC 8270	10	10		<10-<13	NA	<10	NA	NA	NA	<10	NA	<10
Pesticides	0.02-1	0.2-10		<0.2J-<25J	NA	<0.2J-<10J	NA	NA	NA	<0.2J-<10J	NA	<0.01J-<0.5J
TOC	5,000	5,000		7,480	NA	24,100	NA	NA	NA	<5,000	NA	<5,000
TSS	100	200		7,000-35,000	NA	18,000	NA	NA	NA	NA	NA	<100
TDS	10,000	10,000		91,000-151,000	NA	457,000	NA	NA	NA	NA	NA	<10,000

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-8. VEHICLE STORAGE AREA ANALYTICAL DATA SUMMARY

Installation: Wainwright Matrix: Soil/Sediment
Site: Vehicle Storage Area (SS09) Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples							Field Blanks			Lab Blanks	
					S01-2.5	S02-2.5	S03-4	S04-0.75	S05-0.75	S06	SD01	AB01	EB02	TB02		
Laboratory Sample ID Numbers					1492	1494	1496 4483-4	1502	1504	1506 4483-8	1490	1424	1498/1500	1422 4482-6	#5-9193 #1&2-9293 4482	#5-9193 #3&4-9493 #3&4-9293 4483
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	5-33	50-330	500[a]	<50[b]-<300J[b]	<70[b]	<80[b]	<160[b]	<140[b]	<330[b]	<50[b]	<70[b]	NA	<1,000[b]	NA	<1,000J	<200
GRPH	0.2-2	2-20	100	<2J[b]-<5J[b]	<2J[b]	<20J[b]	<20J[b]	<2J[b]	<2J[b]	<2J[b]	<2J[b]	<50J[b]	<50J[b]	<50J[b]	<50J	<2J-<20J
RRPH (Approx.)	11-67	110-670	2,000[a]	<100-<800	<140	<180	<320	<280	<670	<110	<130	NA	<2,000	NA	<2,000	<2,000
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1-<0.5	<1.0J	<1.0	<1.0J	<1.0J	<1.0J	<1.0J	<1.0J					
Benzene	0.02	0.2	0.5	<0.02-<0.1	<0.2J	<0.2	<0.2J	<0.2J	<0.2J	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2J
Toluene	0.02	0.2		<0.02-<0.1	<0.2J	<0.2	<0.2J	<0.2J	<0.2J	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2J
Ethylbenzene	0.02	0.2		<0.02-<0.1	<0.2J	<0.2	<0.2J	<0.2J	<0.2J	<0.2J	<0.2J	<1	<1	<1	<1	<0.02-<0.2J
Xylenes (Total)	0.04	0.4		<0.04-<0.2	<0.4J	<0.4	<0.4J	<0.4J	<0.4J	<0.4J	<0.4J	<2	<2	<2	<2	<0.04-<0.4J
HVOC 8010	0.002	0.02		<0.02-<0.5	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<1	<1	<1	NA	<0.02
VOC 8260																
Tetrachloroethene	0.020	0.050-0.100		<0.020-<0.400	NA	NA	0.330	NA	NA	<0.100	NA	NA	NA	<1	<1	<0.020
Toluene	0.020	0.050-0.100		<0.020-<0.400	NA	NA	0.172	NA	NA	<0.100	NA	NA	NA	<1	<1	<0.020
Trichloroethane	0.020	0.050-0.100		<0.020-<0.400	NA	NA	0.062	NA	NA	<0.100	NA	NA	NA	<1	<1	<0.020
PCBs	0.01-0.07	0.1-0.7	10	<0.1-<2.8J	<0.1	<0.1	<0.3	<0.3	<0.7	<0.1	<0.1	NA	<2	NA	<2	<2

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.

TABLE G-8. VEHICLE STORAGE AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Vehicle Storage Area (SS09)[c]
Matrix: Soil/Sediment
Units: mg/kg

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples				Field Blanks			Lab Blanks	
					2S07-1.5	2SD02	STKP-S01	STKP-S02	AB01	2EB03	2TB03		
Laboratory Sample ID Numbers					1898	1884	1900 4695-4	1902 4695-5	1424	1894/1896 4695-3	1886 4694-9	#6-9993 #1&2-9793 #1&2-9293 4695/4694	#6-91093 #1&2-91093 4695
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mgkg	µg/L	µg/L	µg/L	µg/L	mg/kg
DRPH	5-10	50-100	500[a]	<50[b]-<300J[b]	<100J[b]	<50J[b]	<60J[b]	<60J[b]	NA	<1,000J[b]	NA	<1,000	<50
GRPH	0.1-0.2	1-2	100	<2J[b]-<5J[b]	<2J[b]	<1J[b]	<1J[b]	<1J[b]	<50J[b]	<50J[b]	<50J[b]	<2J-<50J	<1J
RRPH (Approx.)	12-20	120-200	2,000[a]	<100-<600	<200	<120	<140	<140	NA	<2,000	NA	<2,000	<100
BTEX (8020/8020 Mod.)			10 Total BTEX	<0.1-<0.5	<0.20	<0.10	<0.10	<0.10					
Benzene	0.002-0.004	0.02-0.04	0.5	<0.02-<0.1	<0.04	<0.02	<0.02	<0.02	<1	<1	<1	<1-<5	<0.02
Toluene	0.002-0.004	0.02-0.04		<0.02-<0.1	<0.04	<0.02	<0.02	<0.02	<1	<1	<1	<1	<0.02
Ethylbenzene	0.002-0.004	0.02-0.04		<0.02-<0.1	<0.04	<0.02	<0.02	<0.02	<1	<1	<1	<1	<0.02
Xylenes (Total)	0.004-0.008	0.04-0.08		<0.04-<0.2	<0.08	<0.04	<0.04	<0.04	<2	<2	<2	<2	<0.04
HVOC 8010	0.01-0.02	0.1-0.2		<0.02-<0.5	<0.2J	<0.1J	<0.1J	<0.1J	<1	<5	<5	<1J-<10J	<0.02J
VOC 8260													
Naphthalene	0.020	0.020-0.025		<0.020-<0.400	NA	NA	0.053J	0.072	NA	<1	<1	<1	<0.020
Toluene	0.020	0.020-0.025		<0.020-<0.400	NA	NA	<0.025J	0.027	NA	<1	<1	<1	<0.020
1,2,4-Trimethylbenzene	0.020	0.020-0.025		<0.020-<0.400	NA	NA	0.028J	0.042	NA	<1	<1	<1	<0.020
Xylenes (Total)	0.040	0.040-0.050		<0.040-<0.800	NA	NA	0.031J	0.125	NA	<2	<2	<2	<0.040
SVOC 8270													
Benzyl alcohol	0.200	0.220-0.230		<0.200-32.0	NA	NA	<0.230	0.694	NA	<21	NA	<10	<0.200
TOC				10,500-44,100	NA	NA	9,880	6,780	NA	NA	NA	NA	NA

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
a The action levels for DRPH and RRPH are based on conversations with ADEC; final action levels have not yet been determined.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.
c Stockpiled soils (STKP) located in the vicinity of the Vehicle Storage Area were sampled in conjunction with this site.

TABLE G-8. VEHICLE STORAGE AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Vehicle Storage Area (SS09)[c]
Matrix: Soil
Units: mg/kg
METALS ANALYSES

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples				Field Blanks		Lab Blanks
					S03-4	S06	STKP-S01	STKP-S02	EB02	2EB03	
Laboratory Sample ID Numbers					4483-4	4483-8	4695-4	4695-5	4483-9	4695-3	4695 4483
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L
Aluminum	0.35	2		1,500-25,000	1,450	1,200	2,800	2,800	<100	<100	<100
Antimony	N/A	37-52		<7.8-<230	<37	<51J	<47	<52	<100	<100	<100
Arsenic	0.11	5.1-52		<4.9-8.5	<37	<5.1	<47	<52	<100	<100	<100
Barium	0.024	1		27-390	170	59J	100	160	<50	<50	<50
Beryllium	N/A	2.5-26		<2.6-6.4	<18	<2.5	<23	<26	<50	<50	<50
Cadmium	0.33	2.5-18		<3.0-<36	<18	<2.5	<2.3	<2.6	<50	<50	<50
Calcium	0.69	4		360-59,000	1,500	1,700	3,800	3,100J	<200	<200	<200
Chromium	0.066	1		<4.3-47	7.6	5.3	8.4	11	<50	<50	<50
Cobalt	N/A	1-5.2		<5.1-12	3.8	<5.1	<4.7	<5.2	<100	<100	<100
Copper	0.045	1		<2.7-45	4.8	9.4	5.9	8.0	<50	<50	<50
Iron	0.50	2		5,400-35,000	24,400	12,300	17,000	16,000	<100	<100	<100
Lead	0.13	4.7-36		<5.1-22	<36	<5.1	<4.7	<5.2	<100	<100	<100
Magnesium	0.96	4		360-7,400	800	1,060J	1,800	1,700	<200	<200	<200
Manganese	0.025	1		25-290	230	150J	168	150	<50	<50	<50
Molybdenum	N/A	1.8-2.6		<2.5-<11	<1.8	<2.5	<2.3	<2.6	<50	<50	<50
Nickel	0.11	1		4.2-46	11	5.5	10	10	<50	<50	<50

☐ CT&E Data.
N/A Not available.
J Result is an estimate.
c Stockpiled soils (STKP) located in the vicinity of the Vehicle Storage Area were sampled in conjunction with this site.

TABLE G-8. VEHICLE STORAGE AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Vehicle Storage Area (SS09)[c]
Matrix: Soil
Units: mg/kg

METALS ANALYSES

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples				Field Blanks		Lab Blanks
					S03-4	S06	STKP-S01	STKP-S02	EB02	2EB03	
Laboratory Sample ID Numbers					4483-4	4483-8	4695-4	4695-5	4483-9	4695-3	4695 4483
ANALYSES	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	µg/L	µg/L	µg/L
Potassium	23	100		<300-2,200	230	430J	610	690J	<5,000	<5,000	<5,000
Selenium	1.2	36-52		<7.8-<170	<36	<51	<47	<52	<100	<100	<100
Silver	0.53	18-26		<3-<110	<18	<25R	<23	<26J	<50	<50	<50
Sodium	0.55	5		<160-680	170	52	140	290J	<250	<250	<250
Thallium	0.011	0.18-0.25		<0.2-<1.2	<0.18	<0.24	<0.25	<0.24	<5	<5	<5
Vanadium	0.036	1		6.3-59	15	9.0	16	17	<50	<50	<50
Zinc	0.16	1		9.2-95	23	32	22	22	<50	<50	<50

☐ CT&E Data.
J Result is an estimate.
R Result has been rejected.
c Stockpiled soils (STKP) located in the vicinity of the Vehicle Storage Area were sampled in conjunction with this site.

TABLE G-8. VEHICLE STORAGE AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright Matrix: Surface Water
Site: Vehicle Storage Area (SS09) Units: µg/L

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples		Field Blanks					Lab Blanks
					SW01	2SW02	AB01	EB02	TB02	2EB03	2TB03	
Laboratory Sample ID Numbers					1444/1446 4483-10	1880/1882 4694-5	1424	1498/1500 4483-9	1422 4482-6	1894/1896 4695-3	1886 4694-9	#6-9993 #5-9493 #5-9193 #1&2-9793 #1&2-9293 4695/4694 4483/4482
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
DRPH	100	1,000		<1,000J[b]	<1,000[b]	<1,000J[b]	NA	<1,000[b]	NA	<1,000J[b]	NA	<200-<1,000J
GRPH	5	50		<50J[b]-<100J[b]	<50J[b]	<50J[b]	<50[b]	<50J[b]	<50J[b]	<50J[b]	<50J[b]	<2J-<50J
RRPH (Approx.)	200	2,000		<2,000	<2,000	<2,000	NA	<2,000	NA	<2,000	NA	<2,000
BTEX (8020/8020 Mod.)												
Benzene	0.1	1	5	<1	<1	<1	<1	<1	<1	<1	<1	<1
Toluene	0.1	1	1,000	<1	<1	<1	<1	<1	<1	<1	<1	<1
Ethylbenzene	0.1	1	700	<1	<1	<1	<1	<1	<1	<1	<1	<1
Xylenes (Total)	0.2	2	10,000	<2	<2	<2	<2	<2	<2	<2	<2	<2
HVOC 8010	0.5-1	5-10		<5-<10	<1	<5	<1	<1	<1	<5	<5	<1J-<10J
VOC 8260												
1,2-Dichloroethane	1	1	5	<1	1.6	<1	NA	NA	<1	<1	<1	<1
SVOC 8270	10	10-11		<10-<13	<10	<11[c]	NA	NA	NA	<21	NA	<10

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.
b DRPH and GRPH concentrations reported for these samples are equivalent to diesel and gasoline range organics (DRO and GRO) as defined by ADEC.
c The laborabory reported that a possible error occurred during extraction process associated with this sample which resulted in no recoveries for phenolic surrogate and spike compounds.

TABLE G-8. VEHICLE STORAGE AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright Matrix: Surface Water
Site: Vehicle Storage Area (SS09) Units: µg/L

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Levels	Environmental Samples		Field Blanks					Lab Blanks
					SW01	2SW02	AB01	EB02	TB02	2EB03	2TB03	
Laboratory Sample ID Numbers					1444/1446 4483-10	1880/1882 4694-5	1424	1498/1500 4483-9	1422 4482-6	1894/1896 4695-3	1886 4694-9	#6-9993 #5-9493 #5-9193 4695/4483
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
Pesticides	0.001-2.5	0.01-25		<0.2J-<25J	<0.01-<0.2	<0.2J-<25J	NA	<0.2J-<25J	NA	<0.2J-<25J	NA	<0.2J-<10J
PCBs	0.2	2	0.5	<2J	<2J	<2J	NA	<2	NA	<2J	NA	<2J
TOC	5,000	5,000		7,480	107,000	NA	NA	5,000	NA	<5,000	NA	<5,000
TSS	100	200		7,000-35,000	750,000	15,000	NA	NA	NA	NA	NA	<100
TDS	10,000	10,000		91,000-151,000	587,000J	425,000J	NA	NA	NA	NA	NA	<10,000

☐ CT&E Data.
▩ F&B Data.
NA Not analyzed.
J Result is an estimate.

TABLE G-8. VEHICLE STORAGE AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright
Site: Vehicle Storage Area (SS09)
Matrix: Surface Water
Units: µg/L
METALS ANALYSES: TOTAL (DISSOLVED)

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples						Field Blanks		Lab Blanks
					SW01	SW02					EB02	2EB03	
Laboratory Sample ID Numbers					4483-10	4694-5					4483-9	4695-3	4483 4694 4695
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L					µg/L	µg/L	µg/L
Aluminum	17.4	100		<100-350 (<100-340)	9,700 (<100)	<100 (<100)					<100	<100	<100
Antimony	N/A	100	6	<100 (<100)	<100 (<100)	<100 (<100)					<100	<100	<100
Arsenic	5.3	100	50	<100 (<100)	<100 (<100)	<100 (<100)					<100	<100	<100
Barium	1.2	50	2,000	<50-93 (<50-91)	750 (220)	200 (140)					<50	<50	<50
Beryllium	N/A	50	4	<50 (<50)	<50 (<50)	<50 (<50)					<50	<50	<50
Cadmium	1.7	50	5	<50 (<50)	<50 (<50)	<50 (<50)					<50	<50	<50
Calcium	34.5	200		4,500-88,000 (4,100-86,000)	71,000 (57,000)	40,000 (40,000)					<200	<200	<200
Chromium	3.29	50	100	<50 (<50)	<50 (<50)	<50 (<50)					<50	<50	<50
Cobalt	N/A	100		<100 (<100)	<100 (<100)	<100 (<100)					<100	<100	<100
Copper	2.3	50	1,300	<50 (<50)	<50 (<50)	<50 (<50)					<50	<50	<50
Iron	25	100		180-2,800 <100-1,600)	130,000 (17,000)	12,000 (250)					<100	<100	<100
Lead	6.6	100	15	<100 (<100)	<100 (<100)	<100 (<100)					<100	<100	<100

☐ CT&E Data.
N/A Not analyzed.

TABLE G-8. VEHICLE STORAGE AREA ANALYTICAL DATA SUMMARY (CONTINUED)

Installation: Wainwright Matrix: Surface Water METALS ANALYSES: TOTAL (DISSOLVED)
Site: Vehicle Storage Area (SS09) Units: µg/L

Parameters	Detect. Limits	Quant. Limits	Action Levels	Bkgd. Range from 7 DEW Line Installations	Environmental Samples						Field Blanks		Lab Blanks
					SW01	SW02					EB02	2EB03	
Laboratory Sample ID Numbers					4483-10	4694-5					4483-9	4695-3	4483 4694 4695
ANALYSES	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L					µg/L	µg/L	µg/L
Magnesium	47.8	200		<5,000-53,000 (2,600-54,000)	57,000 (48,000)	49,000 (48,000)					<200	<200	<200
Manganese	1.24	50		<50-510 (<50-120)	3,800 (3,000)	130 (70)					<50	<50	<50
Molybdenum	N/A	50-100		<50 (<50)	<50 (<50)	<100R (<50R)					<50	<50	<50
Nickel	5.5	50	100	<50 (<50)	51 (<50)	<50 (<50)					<50	<50	<50
Potassium	1,154	5,000		<5,000 (<5,000)	<5,000 (<5,000)	<5,000 (<5,000)					<5,000	<5,000	<5,000
Selenium	62.4	100	50	<100 (<100)	<100 (<100)	<100 (<100)					<100	<100	<100
Silver	2.6	50	50	<50 (<50)	<50 (<50)	<50 (<50)					<50	<50	<50
Sodium	27.7	250		8,400-410,000 (8,200-450,000)	27,000 (27,000)	45,000 (45,000)					<250	370	<250
Thallium	0.57	5	2	<5 (<5)	<5 (<5)	<5 (<5)					<5	<5	<5
Vanadium	1.8	50		<50 (<50)	63 (<50)	<50 (<50)					<50	<50	<50
Zinc	8.2	50		<50-160 (<50)	3,300 (390)	<50 (<50)					<50	<50	<50

☐ CT&E Data.
N/A Not analyzed.
R Result has been rejected.